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VAN DIE REDAKSIE

BELUISTERING BY MYTERKLEPVERNOUING

Van al die prosedures wat by die moderne diagnose van hartkwale beoefen word, is beluistering die waardevolste. Myterklepvernouing is 'n goeie voorbeeld van die waarde van hierdie tegniek, want hier word noukeurige beluistering met 'n magdom van inligting beloon.

By 'n tipiese geval van 'n baie nou stenose (sonder enige komplikasies) van die myterklep, sal beluistering by die hartpunt in elke siklus 5 liggaamlike tekens uitlok. Hierdie tekens kom in die volgende orde voor: 'n voor-sistoliese geruis; 'n harde klap as 1ste hartklank; 'n normale 2de klank; 'n oopgaan-klapgeluid; en 'n lae, rammelende middel-diastoliese geluid. Hierdie volgorde is in 1862[1] deur Duroziez uitgeken, toe hy dit foneties as ‚ffout-tatarou' beskryf het. ‚Ffout' is die voor-sistoliese geluid wat aangroei tot 'n harde 1ste hartklank; ‚tata' is die 2de klank waarop die oopgaan-klapgeluid volg; en ‚rou' is die middel-diastoliese geluid. Elke onderdeel van hierdie klassieke vyftal word op sy beurt bespreek.

Die *voor-sistoliese geluid* word veroorsaak wanneer die oorvergrote linker-voorkamer teen die einde van die ventrikulêre diastool saamtrek en die bloed deur die vernoude myteropening forseer. Dit is dus gedemp by voorkamertrilling, en word nie gedurende vroegtydige kamersistool gehoor nie. As daar ernstige hartversaking is, kan die vergrote linker-voorkamer nie die bloed sterk genoeg stoot om 'n hoorbare effek te hê nie, en dan verdwyn die voor-sistoliese geluid. Hierdie geluid verskyn ook nie wanneer kwaai drukverhoging in die longslagaar die bloedstuwing deur die linkerhart drasties verminder nie.[2] As 'n eersterangse hartblokkade teenwoordig is, kan hierdie geluid geskei word van die 1ste hartgeluid[3] en ietwat vroeër in die diastool voorkom. By algehele hartblokkade, met totale afsluiting tussen die voor- en die hartkamer, kom voorkamer-sistoolgeluide nog voor, maar hulle het dan geen konstante betrekking op die hartklanke nie.

Die *harde 1ste hartklank* ontstaan op die volgende manier: Aan die einde van diastool forseer die hoë druk in die linker-voorkamer die slippe van die klep diep in die linkerkamer in. Die daaropvolgende skielike sametrekking van die kamer druk die klepslippe teen

EDITORIAL

AUSCULTATION IN MITRAL STENOSIS

The most useful single procedure in modern cardiological diagnosis is auscultation. Its value is well illustrated by considering mitral stenosis, in which diligent auscultation is rewarded by a wealth of information.

In a typical case of uncomplicated, tight mitral stenosis, auscultation at the cardiac apex will elicit 5 physical signs in each cycle. These are, in sequence, a presystolic murmur, a loud, snapping 1st heart-sound, a normal 2nd sound, an opening snap, and a low-pitched, rumbling mid-diastolic murmur. This sequence was recognized by Duroziez in 1862,[1] when he characterized it phonetically as 'ffout-tatarou'. 'Ffout' is the presystolic murmur, rising in crescendo to a sharp 1st heart-sound; 'tata' is the 2nd sound followed by the opening snap, and 'rou' is the mid-diastolic rumble. Each component of this classical pentalogy will be considered in turn.

The *presystolic murmur* is produced by the hypertrophied left auricle when it contracts towards the end of ventricular diastole and forces blood through the narrowed mitral orifice. It is therefore abolished by auricular fibrillation and is not heard during ventricular premature systoles. When there is gross cardiac failure, the dilated left auricle is incapable of propelling the blood with sufficient force to produce an audible effect, and the presystolic murmur disappears. The murmur is also abolished when severe pulmonary hypertension drastically reduces the blood flow through the left heart.[2] In the presence of 1st-degree heart-block (i.e. prolonged auriculo-ventricular conduction) the murmur may be separated from the 1st heart-sound[3] and assume a position somewhat earlier in diastole. In complete heart-block, with total dissociation of auricular and ventricular activity, auricular systolic murmurs still occur but bear no constant relation to the heart sounds.

The *loud 1st heart-sound* is produced as follows: The high left auricular pressure forces the cusps of the mitral

mekaar, en omdat hulle ietwat verstyf is, gaan hulle met 'n kortaf klapgeluid toe—die ‚toegaan-klapgeluid van die myterklep'.[4] Hierdie teken toon dus aan dat die druk in die linker-voorkamer verhoog is, en dat die klepslippe, nieteenstaande hul ligte fibrose, nog soepel is met betreklik vry chordae tendineae. In so 'n geval behoort dit maklik te wees om die verbinding met die vingers te skei.[4] Die 1ste geluid verloor hierdie hoedanighede as die klep weens hewige fibrose of verkalking heeltemal styf word, en as die chordae tendineae baie littekens dra. Onder hierdie omstandighede kan 'n tegnies moeilike valvotomie verwag word. By eersterangse hartblokkade, waar die klepslippe by die aanvang betreklik naby mekaar is, en by myterklepverswakking, waar hulle nie behoorlik kan toegaan nie, is die 1ste klank kenmerkend sag. Aktiewe hartontsteking is geneig om die 1ste geluid te demp, en by voorkamertrilling varieer die sterkte daarvan volgens die kringloop van die hartslag.

Die 2*de hartgeluid* word veroorsaak deur die toegaan van die halfmaankleppe. Die 2de of longslagaarklepgedeelte van hierdie geluid word harder namate die druk in die longslagaar styg. By myterklepvernouing is die tweede geluid nie abnormaal verdeel nie; 'n lang tussenpose tussen die samestellende klanke beteken dat daar belemmering in His se bondel is.

Die *oopgaan-klapgeluid van die myterklep*[5] kom omtrent 0·08 sekondes na die aanvang van die 2de geluid voor. Dit ontstaan wanneer die ietwat verstyfde klepslippe oopgegooi word deur die vinnige instroming van bloed in die linkerkamer vroeg in die diastool. Dit is 'n hoë klank, en hoewel dit die beste gehoor kan word by die onderste rand van die borsbeen of by die hartpunt, is dit dikwels wyd versprei oor die hele hartstreek. Dit is 'n waardevolle teken omdat dit blykbaar kenmerkend is van myterklepvernouing, en omdat dit nie voorkom by aandoenings wat myterklepvernouing naboots met middel-diastoliese en voor-sistoliese geluide nie. Dit is dus afwesig by ventrikulêre tussenskotdefek, by oop ductus arteriosus, skildkliervergiftiging, bloedarmoede, en by 'n gewas in die linkervoorkamer. Ook vergesel dit nie die Austin Flint-geluid nie. As hierdie teken wel voorkom, beteken dit gevestigde myterklepstenose met soepel klepslippe en minimale terugvloeiing. Net soos die harde 1ste klank, kom dit nie voor as die klepslippe vol littekens om met kalk aangeslaan is nie.

Die *rammelende middel-diastoliese geluid* word veroorsaak as die bloed uit die voorkamer deur die vernoude myterklep in die kamer vloei. Die klank word die beste opgeneem met 'n ‚klok'-tipe gehoorpyp net by die hartpunt met die pasiënt links gedraai. Liggaamsoefening kan dit beklemtoon. Die Valsalva-maneuwer kan hierdie geluid uitwis,[2] en dikwels word dit uitgeskakel by groot hartversaking of by hoë druk in die longslagaar.[3] Dit is nie waarneembaar by gevalle waar die myterklep aansienlik onbekwaam is nie.

Benewens hierdie 5 tekens, kan sekere *bykomende* tekens onder bepaalde omstandighede ontwikkel. As ernstige drukverhoging in die longslagaar ontwikkel, sit hierdie slagaar uit, en dan kan 'n vroeë sistoliese klikgeluid[7] in die longstreek gehoor word. Die buitengewoon hoë druk in die longslagaar kan terugvloeiing by die longslagaarklep veroorsaak, as gevolg waarvan

valve deep into the left ventricle at the end of diastole. Then the ventricle contracts, suddenly forcing the cusps together and, because of their slight rigidity, they close with an abrupt snap—'the closing snap of the mitral valve'.[4] This sign thus indicates that the left auricular pressure is elevated and that the cusps, though somewhat fibrotic, are still pliant, with relatively free chordae tendineae. In such a case digital separation of the commissure should be readily accomplished.[4] The 1st sound loses these qualities if the valve is made rigid by excessive fibrosis or calcification or if there is much cicatrization of the chordae tendineae; in these circumstances a technically difficult valvotomy must be anticipated. In 1st-degree heart-block, where the cusps are relatively close together at the onset of systole, and in mitral incompetence, where they cannot shut properly, the 1st sound is characteristically soft. Active carditis tends to subdue the 1st sound and in auricular fibrillation its intensity will vary with cycle length.

The 2*nd heart-sound* is produced by closure of the semilunar valves. The 2nd or pulmonary-valve component of this sound increases in intensity as the pressure in the pulmonary artery increases. The 2nd sound is not abnormally split in mitral stenosis; wide separation of its components indicates bundle branch block.

The *opening snap of the mitral valve*[5] occurs about 0·08 seconds after the beginning of the 2nd sound. It is produced when the somewhat stiffened cusps are flung open by the rapid inflow of blood into the left ventricle during early diastole. It is a high-pitched sound and although best heard at the lower left sternal border or at the cardiac apex, it is often widely conducted over the praecordium. It is a valuable sign because it seems to be pathognomonic of mitral stenosis, and does not occur in conditions which simulate mitral stenosis by producing mid-diastolic and presystolic murmurs. Thus, it is absent in ventricular septal defect, patent ductus arteriosus, thyrotoxicosis, anaemia and left auricular tumour, and does not accompany the murmur of Austin Flint. Its presence indicates established mitral stenosis with pliant cusps and minimal regurgitation. Like the loud 1st sound, it is absent when the cusps are heavily scarred or plastered with calcium.[6]

The *mid-diastolic, rumbling murmur* is produced by the flow of blood from auricle to ventricle through the stenosed mitral orifice. It is best heard through a bell-type stethescope just medial to the apex, with the patient inclined to his left side. It may be accentuated by exercise. The Valsalva manoeuvre may obliterate this murmur[2] and it is often abolished by gross cardiac failure or by severe pulmonary hypertension.[3] It is not heard in cases with considerable mitral incompetence.

In addition to these 5 signs, certain *additional* signs may develop under special circumstances. When severe pulmonary hypertension develops, the pulmonary artery dilates and an early systolic click[7] may be heard in the pul-

Graham Steell se vroeë, blasende diastoolgeluid voorkom. Die 3de hartgeluid is nie opvallend by myterklepvernouing sonder komplikasies nie; as dit voorkom, beteken dit moontlik aktiewe ontsteking in die hart of onbekwaamheid van die myterklep. Dit kan onderskei word van die oopgaan-klapgeluid omdat dit laer gestem is en baie later in die diastool voorkom. In so 'n geval is myter-valvotomie nie raadsaam nie.[3]

Ten laaste kan daar 'n *sistoliese geluid* wees wat groot versigtigheid by vertolking verg. As dit sag is en slegs tot 'n gedeelte van die sistool beperk is, is dit waarskynlik van min belang. As dit egter gedurende die hele sistool en selfs verby die hartpunt hoorbaar is, dui dit myterklep-onbekwaamheid aan.[8] By 'n nou stenose van die myterklep kan sommige pansistoliese geluide ook by die hartpunt voorkom; hulle ontstaan gewoonlik as die drieslippige kleppe weens verhoogde druk in die longslagaar gebrekkig funksioneer. Hierdie geluide word kenmerkend harder by inaseming[9] en word vergesel van voelbare oorvergroting van die regterkamer, sistoliese polsing in die lewer, en 'n merkbare sistoolgolf in die nek-aarpols. So 'n sistoliese geluid is nie 'n teenaanwysing vir 'n myterklep-operasie nie—dit verg dit eerder.

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4. Holmes Sellors, T., Evan Bedford, D. en Somerville, W. (1953): *Ibid.*, **2**, 1059.
5. Mounsey, P. (1953): Brit. Heart J., **15**, 135.
6. Wynn, A. (1953): *Ibid.*, **15**, 214.
7. Leatham, A. en Vogelpoel, L. A. (1954): *Ibid.*, **16**, 21.
8. Brigden, W. en Leatham, A. (1953): *Ibid.*, **15**, 55.
9. Muller, O. en Shillingford, J. (1954): *Ibid.*, **16**, 195.

monary area. The unusually high pressure in the pulmonary artery may produce regurgitation at the pulmonary valve, resulting in the early, blowing diastolic murmur of Graham Steell. The 3rd heart-sound is not obvious in uncomplicated mitral stenosis; its occurrence suggests active carditis or mitral incompetence, and it may be distinguished from the opening snap by its lower pitch and because it occurs much later in diastole. In its presence, mitral valvotomy is generally not advisable.[3]

Finally, there may be a *systolic murmur*, and its interpretation requires careful judgment. If it is soft and confined to part of systole only, it is probably of little consequence. If, on the other hand, it occupies all of systole and is conducted beyond the apex, mitral incompetence is indicated.[8] Some loud pansystolic murmurs may, however, occur at the apex in tight mitral stenosis; these usually arise from tricuspid valves which are functionally incompetent because of pulmonary hypertension. Such murmurs characteristically increase with inspiration[9] and are accompanied by palpable right ventricular hypertrophy, by systolic pulsation of the liver, and by a prominent systolic wave in the jugular venous pulse. Such a systolic murmur does not contra-indicate mitral valvotomy, but rather calls for it.

1. Duroziez, P. (1862): Quoted by Ongley *et al.*, *loc. cit.*
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SOME OBSERVATIONS ON LEUKAEMIA: A PRELIMINARY NOTE*

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The nature of the leukaemias or leukoses has been a subject of much discussion ever since the disease was first described by Craigie, and by Bennet, and by Virchow, just a century ago and the problem is far from solved even yet. That this is still true is very apparent from the account of the proceedings of the Ciba Symposium on Leukaemia Research held in November 1953 when, it seems, a common basis for discussion by the various authorities there gathered was difficult to find (*vide loc. cit.* p. 32 *et seq.*)

Various theories have been put forward concerning the nature of these diseases, for example, the infective, nutritional deficiency, and neoplastic theories. The infective theory was in the background so far as mammalian leukaemia was concerned for many years, but has recently been brought to the fore again as a result of the experimental work, in mice, of Gross, who claims to have demonstrated a filter-passing causative agent (Gross, 1954).

So far as the nutritional or maturing-factor deficiency theories are concerned, there is very little evidence in their favour, however attractive they may be as ideas in that they hold out more hope of cure than a neoplastic theory does. The only advocate of this theory who need be taken seriously is Sir Lionel Whitby (1951), but there are many loopholes in his arguments and his views have been criticized by Furth (1951).

The observations submitted here are, of course, tentative and are only put forward as a basis for discussion.

Of the neoplastic nature of human leukaemia I feel there is no doubt. The hypothesis I wish to submit here, which I do not claim as original although I have been unable to find any reference to a similar suggestion elsewhere, is that the chronic leukaemias may be regarded as benign neoplasms and the acute leukaemias as malignant neoplasms. No attempt can be made here to define

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these two terms, benign and malignant, except to give the simplest of examples to show what is meant. A circumscribed, non-metastasizing tumour of, for example, fibrocytes, is a benign fibroma; a metastasizing invasive tumour of fibrous tissue, on the other hand is malignant—a sarcoma. It is also known that the first may change into the second and, further, that these terms are not entirely (although they are usually) synonymous with clinical ideas of benign and malignant. As Ewing many years ago pointed out, 'If malignancy were a purely clinical conception, it would be impossible to draw any rigid distinction between benign and malignant tumors, since nearly all tumors may prove fatal' (Ewing, 1940).

BLOOD AS A TISSUE

Now in considering these concepts certain points must be emphasized. Firstly a stumbling block to acceptance of the neoplastic nature of these diseases that is apparent in the literature is the absence of tumorous deposits. But a fundamental fact which must be borne in mind is the concept probably universally accepted, but not emphasized sufficiently, that the blood constitutes a fluid tissue not separate from but co-terminous with the blood-forming organs, whether bone marrow, lymph-nodes or spleen. Boycott introduced this idea with his term the 'erythron' (Boycott, 1929) for the red cells and their precursors, but an even wider term is required.

Regarding the blood, then as a fluid tissue, it need not surprise that a neoplastic condition of one of its elements does not always result in actual tumours, and that the effects of such a neoplasm, although benign in the sense in which it is here used, may nevertheless have far-reaching effects.

The chronic leukaemias are characterized by a proliferation of essentially mature cell types, which have their identical counterparts in the normal. Much time has been spent endeavouring to demonstrate a biochemical or cytological deviation from these normal counterparts, but it may be fairly said that no convincing difference has so far been demonstrated, and this is as might be expected of the cells of a benign neoplasm. As regards their invasive properties, it is agreed by histopathologists that the cells of the chronic leukaemias have little if any, true invasive power, their presence in all organs and tissues being merely an expression of the ubiquity of blood as a tissue.

The course, too, of the chronic leukaemias, suggests a benign neoplasm, but here a change appears to have occurred in recent times, and it is this change which has led to the development of this hypothesis. Previously, it appears, the usual termination of the chronic leukaemias was for the patient to succumb to an intercurrent infection or a thrombotic episode or to die from anaemia and exhaustion. The possibility of termination as a typical acute leukaemia is mentioned in the older texts, but it is clearly indicated that this was regarded as a rather rare event. Shimkin *et al.* (1951) state that one quarter of the cases of chronic myeloid leukaemia they studied over a 40 year period, died in a terminal acute phase, but these were *post mortem* diagnoses. What does more recent experience show? Out of 19 cases of chronic myeloid leukaemia observed by us until demise, in the past 3 years, no less than 15 (over 75%) terminated as an acute myeloblastic leukaemia, both clinically and haematologically. Of the 4 others, 1 died of coronary thrombosis, 1 of post-operative infection, and 2 of anaemia and exhaustion.

In considering the reasons for this, the possibility that therapeutic measures may be increasing the likelihood of malignant change has been considered. Haddow and others have shown that most of our therapeutic agents are carcinogenic e.g. X-rays, nitrogen mustards, the epoxides, 'myleran', urethane, and others (Haddow, 1953). The following table shows the various agents used to treat these cases, but no special significance can be attached to the figures.

DXT alone	5
DXT + Myleran	7
Myleran alone	2
Urethane alone	1

The course of chronic lymphatic leukaemia is somewhat different from that of chronic myeloid leukaemia in that the prognosis is better and it but rarely terminates as an acute leukaemia. Thus Shimkin found an acute termination in only 2 out of 137 cases of chronic lymphatic leukaemia (Shimkin *et al.*, 1953). Nevertheless I think there is evidence from our material that the character of the disease does sometimes change from a benign to a malignant neoplasia terminally.

It is unusual to perform bone-marrow aspiration biopsies on terminal cases of chronic lymphatic leukaemia, but we have done so on 3 cases. In each case there was what is termed in the American literature 'marrow block'—a state in which the bone-marrow is crowded with cells so that aspiration is difficult and results in only a drop or two of material. This material in the cases studied did not consist of mature lymphocytes such as were still present in the peripheral blood, but was almost entirely primitive lymphoid reticulum cells. Now 2 of these 3 cases had received T.E.M. as treatment at 2 months and 3 months before death, and the 3rd case 2 courses of nitrogen-mustard therapy 6 months and 3 months before death. There was no clue from the peripheral blood that invasion of the marrow by primitive cells had occurred, there being merely severe anaemia, thrombocytopenia and the leucocyte picture of a chronic lymphatic leukaemia. The fact that they do not terminate as acute 'lymphoblastic' leukaemia gives some support to the view held by Naegeli, Rohr and Moeschlin, with which I am in agreement, that acute lymphoblastic leukaemia has no existence in fact, unless possibly during childhood.

A further point which may have some bearing on the apparently infrequent acute termination to chronic lymphatic leukaemia is the fact that this condition is more often treated conservatively than chronic myeloid leukaemia, with usually only sufficient radiation to cause the lymph nodes to regress.

These cases lend some support to the hypothesis here put forward, but clearly further investigation of chronic lymphatic leukaemia in its final stadium must be carried out.

Finally, a concept which has arisen of late years helps I think to keep the matter in a proper perspective. I

refer to the concept of the myelo-proliferative diseases which are shown in Fig. 1. These diseases could, I think, be regarded as benign neoplasms, and the grounds for this concept of myeloproliferative disease is the agreed

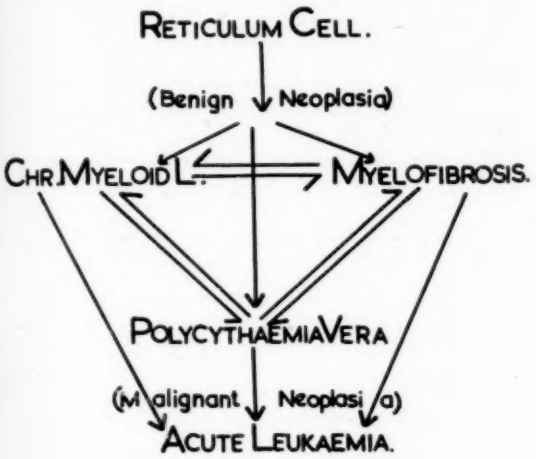

Fig. 1. A schema showing the myelo-proliferative diseases and their interrelations. Chronic myeloid leukaemia, myelofibrosis and polycythaemia vera are regarded as benign neoplasms and acute leukaemia as a malignant neoplasm.

multipotentiality of the primitive reticulum cell, enabling benign neoplasia to occur along the various developmental lines and often along more than one simultaneously, leading to known associations of these conditions. Thus polycythaemia has an association with chronic myeloid leukaemia, and with myelofibrosis. Myelofibrosis may be the end-result of a chronic myeloid leukaemia or of a polycythaemia. Any may terminate as the malignant form, to wit, acute myeloblastic leukaemia. This has been known for some years now to occur more commonly after treatment of polycythaemia with ^{32}P than in untreated cases.

CONCLUSION

To conclude then, the following suggestions are made:

1. Leukaemia is a true neoplasm.

2. The chronic myelogenous and chronic lymphatic forms are benign neoplasms which, on account of the ubiquity of the tissue affected, have far reaching and ultimately fatal effects. (In this sense they are not benign.)

3. Acute myeloblastic leukaemia is the malignant neoplasm corresponding to chronic myelogenous leukaemia, and a lymphoid reticulum cell neoplasm is the malignant neoplasm corresponding to chronic lymphatic leukaemia.

4. This view of the leukaemias is in keeping with the concept of the myeloproliferative diseases.

5. The change from benign to malignant neoplasms is being seen more frequently now as a result of the use of carcinogenic therapeutic agents.

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CHEMOTHERAPY OF LEUKAEMIA*

1. MYLERAN IN THE TREATMENT OF CHRONIC MYELOID LEUKAEMIA

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Rational cancer chemotherapy, according to C. P. Rhoads of the Sloan Kettering Institute for cancer research, has passed through two phases and has entered upon a third.[1]

The first phase was the discovery of agents or procedures causing atrophy of specific tissues and, by virtue of this property, able to influence tumours of that tissue. Examples of this phase are the use of oestrogens in the treatment of cancer of the prostate and radio-active iodine for the treatment of carcinoma of the thyroid. The substance 'Myleran' also belongs to this first category.

The second phase according to Rhoads is the use of compounds injurious to cells in direct ratio to their rate of growth. Examples of these, he suggests, are the antimetabolites of folic acid, aminopterin, etc. The third phase is the use of substances which selectively injure by virtue of the biochemical specificities of the target cells, characteristics which distinguish the neoplastic cell from its normal analogue growing at the same rate. Such a substance he believed was 6-mercapto-purine.

Now while Rhoads described these phases as chrono-

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

logical, I do not think that he would imply that we have by any means exhausted the possibilities of the first two in having perhaps arrived at the third. Indeed, I feel that there is much potential still to be discovered in the first category although I am not very sanguine about the potentialities of the second.

MYLERAN IN THE TREATMENT OF CHRONIC MYELOID LEUKAEMIA

Myleran (1·4 dimethane-sulphonoxybutane) was the outcome of research by Haddow and his associates who found that members of a series with the following type formula shown

$$RN\begin{matrix}\nearrow CH_2CH_2OSO_2R \\ \searrow CH_2CH_2OSO_2R\end{matrix}$$

possessed cytostatic properties for various experimental animal tumours.[2] This finding led them to investigate other substances bearing sulphonic-acid ester groups, and in particular the methane-sulphonoxy-alkanes.[3, 4] The type formula of these substances is as follows:

$$CH_3SO_2O(CH)_nOSO_2CH_3$$

If the value of n lies between 2 to 10, cytostatic activity is found to be present, but maximum activity was found if $n=4$ or 5.

The substance 1·4 dimethane-sulphonoxybutane was found to have a marked inhibitory effect on the Walker rat-carcinoma 265 and a selective depressant effect on myeloid cells both in the rat and man. This effect on the myeloid series is selective, in that doses depressant for that series have no effect on the lymphocyte or erythroid series.[1]

The precise mechanism of the cytostatic action of these compounds is still uncertain,[5] but the substances are bifunctional alkylating agents and may act through formation of carbonium ion ($R.CH_2$) according to the following formula:

$$R.CH_2OSO_2R^1 = R.CH_2^+ + {}^-OSO_2R^1$$

or alternatively, as suggested by Timmis, by the capacity of these compounds to form ring compounds with amino or sulphydryl groups. These substances, in keeping with Haddow's general hypothesis,[5] are carcinogenic, the substance Myleran (1·4 dimethane-sulphonoxybutane) being particularly active in this respect.

Dosage. Myleran is available in 2-mg. and 0·5-mg. sugar-coated tablets. The dosage schedule we followed at first was 2 tablets (4 mg.) of Myleran daily until the blood count showed no further evidence of improvement; i.e., in the majority of cases, when the total leucocyte count had reached normal levels and immature cells had virtually disappeared from the peripheral blood. Concurrently with the improvement in the leucocyte count, and usually more rapidly, the haemoglobin and red-cell count returned to normal values. The length of time a course lasted varied from patient to patient and in some, as the improvement seemed to continue over a period of many months up to a year or more, we were led to try maintenance treatment, when instead of ceasing therapy when maximum benefit has occurred, it is continued at a dose varying from $\frac{1}{4}$ to $\frac{1}{2}$ of the previous treatment level. It has never been found necessary to use a dosage above 4 mg. per day although in some cases doubtless a more rapid response would have been obtained in this way. Continuous therapy at 1-2 mg. per day has been continued throughout the period of observation in some patients.

Results. A total of 34 cases of chronic myeloid leukaemia have been treated with this drug, 22 of whom were Europeans (12 men and 10 women) and 12 Bantu (8 men and 4 women). Of the Europeans 13 are still alive, 8 have died, and information is not available regarding 1. On account of the difficulties of following up the Bantu cases, no general assessment of the results of therapy in them can be made. In all except 3 of the European cases immediate benefit ensued, but it was short-lived (less than 3 months) in 2. The longest remission has been 24 months. Two courses of therapy have been given to 13 Europeans and the response to the second course

TABLE I. EUROPEAN CASES

Case No.	Age (a)	Sex	Previous Treatment	Resistant To DXT	Type of Myleran Treatment	No. of Courses	Response to Courses				Length of Remission (Months)				Alive / Dead
							1	2	3	4	1	2	3	4	
1.	35	F	DXT, many courses	Yes	Courses	2	Good	None	—	—	3	none	—	—	D
2.	30	F	DXT, 2 courses	No	Courses M	2	Good	Good	—	—	7	1 yr. (b)	—	—	A
3.	45	M	DXT ^{32}P	Yes	Maintenance	—	Good	—	—	—	2 yr. (b)	—	—	—	A
4.	45	M	DXT, many courses	Yes	Course	1	Poor	—	—	—	1	—	—	—	D
5.	37	F	DXT, 2 courses	Yes	Maintenance	—	Good	—	—	—	2 yr. (b)	—	—	—	D
6.	52	M	DXT, 2 courses	No	Courses M	4	Good	Good	Good	Good	1	8	10	2 (c)	A
7.	64	F	None	—	Courses M	4	Good	Good	Good	Good	8	6	4	6 (b)	A
8.	34	M	DXT, 2 courses	No	Course	1	Good	—	—	—	2 (c)	—	—	—	A
9.	72	M	TEM. DXT, many courses	Yes	Courses	2	Poor	None	—	—	1	none	—	—	A
10.	24	M	DXT, 2 courses	No	Courses	3	Good	Good	None	—	8	2	none	—	D
11.	40	F	None	—	Courses M	2	Good	Good	—	—	10	14 (b)	—	—	A
12.	48	M	None	—	Courses M	2	Good	Good	—	—	8	1 yr. (b)	—	—	A
13.	49	F	None	—	Courses M	2	Good	Fair	—	—	1 yr.	6 (b)	—	—	D
14.	34	M	DXT, 2 courses	No	Courses	2	Good	None	—	—	8	none	—	—	D
15.	53	F	None	—	Courses	2	Good	Good	—	—	8	4 (c)	—	—	D
16.	60	F	DXT, 4 courses	Yes	Courses	2	Good	None	—	—	3	none	—	—	D
17.	73	M	None	—	Courses	2	Good	Good	—	—	6	4 (c)	—	—	A
18.	55	M	None	—	Maintenance	—	Good	—	—	—	15 (b)	—	—	—	A
19.	49	M	None	—	Course	1	Poor	—	—	—	none	—	—	—	D
20.	67	M	None	—	Course	1	Poor	—	—	—	none	—	—	—	? (d)
21.	38	F	None	—	Maintenance	—	Good	—	—	—	1 (c)	—	—	—	A
22.	43	F	None	—	Maintenance	—	Good	—	—	—	2 (c)	—	—	—	A

DXT = Deep X-ray therapy.
(a) = Age at diagnosis.
(b) = Length of remission on maintenance therapy.
(c) = Remission continuing.
(d) = Patient refused hospital treatment: presumed dead.
Courses M = Initial treatment by courses of Myleran with subsequent assumption of maintenance treatment.

was poor or absent in 5 and good in 8. Seven of the 8 are still alive and a third course has been given with benefit to 2 of them, and without benefit to 1; a fourth course has been given to the same 2 cases. Seven are on continuous dosage and have remained well for periods of 24, 24, 15, 14, 12, 12 and 6 months respectively. Eleven cases had previously received deep X-ray therapy or other source of ionizing radiation, e.g. ^{32}P. Of these only 2 failed to respond to Myleran. Longer remissions as a result of Myleran therapy than were obtained with deep X-ray therapy have been observed in 8 of the 11 cases (Tables I and II).

TABLE II. BANTU CASES

Case No.	*Age*	*Sex*	*Previous Treatment*	*Initial Response to Myleran*	*Notes*
1	30	F	None	Good	In remission from 1 course of 10 mths. Relapse resistant to Myleran. DXT tried without benefit. Became acute and died.
2	26	F	None	Good but short	After 1 mth's. remission became acute in type and died 3 wks. later.
3	45	M	DXT	Fair. Splenomegaly and some anaemia persisted.	No follow-up.
4	23	M	None	None. Given DXT with benefit.	Subsequently required 2 more courses of DXT, became acute and died.
5	?	M	None	Fair. Splenomegaly persisted.	No follow-up.
6	28	M	None	Good.	No follow-up beyond 6 mths., during which time he remained well.
7	40	M	None	Good. Splenomegaly persisted.	No follow-up.
8	42	M	DXT	Good.	Remained well for 10 mths. No further follow-up
9	?	F	None	Poor, after 8 wks. treatment given DXT.	Relapsed after DXT in 2 mths. No further follow-up
10	35	M	None	Good. Splenomegaly persisted.	No follow-up.
11	29	M	None	Good.	Remained well for 4 mths. No further follow-up.
12	30	F	None	Fair. Splenomegaly persisted.	No follow-up. Took own discharge from hospital before treatment was complete.

Toxicity. We have found Myleran to be singularly free from side-effects for a potent cytostatic agent. Bone-marrow depression has been observed once only. Thrombocytopenia has not been observed as a result of therapy, but in two cases thrombocytopenia was present at the start, and in both these cases, the platelet deficiency and haemorrhagic phenomena were aggravated to such extent that therapy had to be discontinued. Minor disturbances due to Myleran have been few; the increase in skin pigmentation noted by Galton[6] and by Petrakis *et al.*,[11] has been observed in 2 cases. Bollag[13] notes amenorrhoea as a complication of Myleran therapy, and menopausal symptoms with amenorrhoea have been observed in 3 female patients in this series.

Termination as acute myeloblastic leukaemia has been observed in 9 cases, 7 of whom received deep X-ray therapy in addition to Myleran and 2 received Myleran only.

DISCUSSION

Galton,[6, 7] Galton and Till,[8] Ledlie[9] and Wilkinson,[10] in Britain, Petrakis *et al.*,[11] and Wintrobe[12] in the USA, Bollag[13] in Switzerland, Hansen[14] in Denmark, Gigante, Teodori and Zoppini[15] in Italy, and Kurrle[16] in Australia, have reported on the use of this drug in cases of chronic myeloid leukaemia.

This literature is uniform regarding the beneficial effects of Myleran therapy in chronic myeloid leukaemia, a situation unusual in the literature relating to a chemotherapeutic agent. All stress the haematological benefit matched by clinical improvement and the remarkable absence of toxic or undesirable side-effects.

In general the results described in this paper are similar to those obtained by the other authors quoted, but several points merit further discussion. It has become apparent that patients kept on maintenance dosage of a low order ($\frac{1}{2}$-1 mg. *per diem*) are maintained more able than those treated with interrupted courses. An important point, however, not to be lost sight of in discussing the relative merits of various forms of treatment, is that all treatment available to date against chronic myeloid leukaemia is palliative only. The best treatment is not necessarily the one which will prolong life longest but the one which will maintain the patient at his fittest in the least disturbing fashion for the remainder of his life. And here Myleran therapy has undoubted advantage over external radiation therapy. All patients who have previously had deep X-ray therapy are unaminous in their preference for Myleran.

We have confirmed the value of myleran therapy in patients in whom radiation therapy is ceasing to have effect. We have also proved its value in cases where deep X-ray therapy could not be carried out on account of the patient's condition.

CONCLUSION

Myleran is an effective, and satisfactory palliative therapy for chronic myeloid leukaemia. Side-effects are very few, and dangerous toxic effects, provided adequate haematological control is exercised, very rare indeed.

I wish to express my thanks to the many physicians and practitioners who have cooperated in the study of these patients; also to Prof. A. Haddow of the Chester Beatty Institute, for generous supply of Myleran.

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CHEMOTHERAPY OF LEUKAEMIA*

II. 6-MERCAPTO-PURINE ('PURINETHOL') IN THE TREATMENT OF ACUTE LEUKAEMIA AND SOME OTHER NEOPLASTIC DISEASES OF THE RETICULOENDOTHELIAL SYSTEM

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6-Mercapto-purine ('Purinethol†) is an example of a chemotherapeutic agent of the third phase of Rhoads.[1] It is an analogue of the nucleic-acid constituent adenine and the physiological purine-base hypoxanthine (Fig. 1) and was one of a very large series of analogues prepared and studied by Hitchings and his colleagues[2] at the Wellcome Research Laboratories. This work was a consideration of the possibility that cells of different characteristics and so having different genes might have desoxyribose-nucleic acid (DNA) of differing

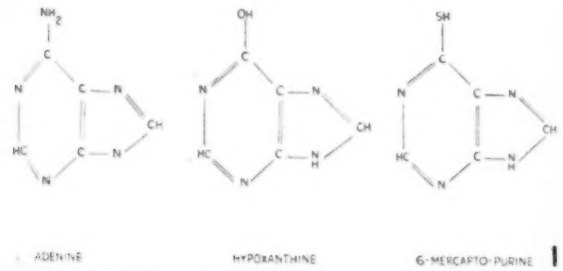


Fig. 1. Structural formula showing relation of 6-mercapto-purine (Purinethol) to adenine and hypoxanthine.

composition in these genes. It was shown conclusively that specificity of DNA does exist and this in turn established a firm foundation for the possibility of developing selectively toxic compounds. 6-Mercapto-purine has been shown to be a purine antagonist for lactobacillus casei, but studies in animals have shown that neither the toxic nor the anti-leukaemic effects can be reversed by simple purines. Indeed, no antidote to its cytotoxic effects in animals has so far been discovered.

Dosage. In patients the drug is generally given at a dosage level of 1 mg. per lb. body-weight, in a single dose daily by mouth. At least 3 weeks of therapy and often up to 6 or 8 weeks are needed before remissions are achieved, and then maintenance therapy at the same or a reduced dose-level is applied. In some cases steroid therapy (cortisone or ACTH) was employed concurrently with Purinethol therapy. Careful haematological control was exercised on the patients, especially during the initial phase of therapy, as the response of any given patient we found to be highly unpredictable. In some, precipitous drops in the leucocyte count occurred; in

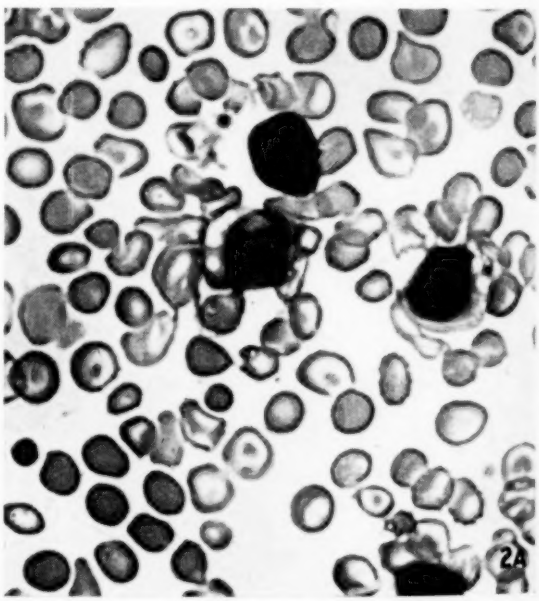


Fig. 2. (*a*) Blood Film: Wrights stain (×620). This shows the marked anisocytosis with macrocytosis of the red cells in a patient after treatment with Purinethol in full doses for 3 months. There is also conspicious target cell formation.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

† Trade name Burroughs Wellcome Ltd.

others prolonged therapy was necessary to effect any alteration in the blood picture.

Toxicity. Leucopenia is usually induced and is, indeed, a prerequisite of successful therapy in most cases. A curious feature noted early in our series was the appearance under treatment of very unusual leucocytes in the peripheral blood, which often defied accurate morphological description. Usually they appeared to be 'monocytoid myelocytes', less commonly 'monocytoid lymphocytes', and their morphology did not seem to depend on the original cytological type of leukaemia. These cells are depicted in Fig. 2.

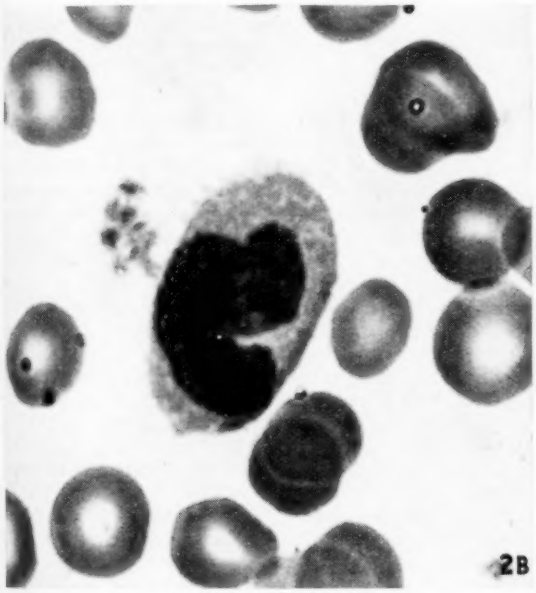


Fig. 2. (*b*) Blood Film: Wrights stain (×1600). Showing an example of the atypical leucocytes which we have regarded as 'monocytoid lymphocytes' which commonly appear in the peripheral blood of patients treated with Purinethol. In staining reactions they closely resemble 'glandular fever' cells.

Thrombocytopenia is rarely seen as a result of therapy; on the contrary, a pre-existing thrombocytopenia often disappears and bleeding phenomena are cured. Irreversible bone-marrow depression attributable to the Purinethol has not been seen. Anaemia has usually not been benefited by the drug, and the appearance of a macrocytic red-cell picture with marked anisopoikilocytosis is common in cases maintained for any period of time on maximum dosage. The bone-marrow in these cases shows partial megaloblastic change of erythropoiesis. In one case, where anaemia was severe and the marrow showed partial megaloblastic changes, folic acid orally was tried without benefit to the anaemia (or detriment to the leukaemia).

Oral lesions and gastro-intestinal symptoms referable to the therapy have not been observed. Marked loss of hair was noted in one patient, but this had commenced prior to the treatment, and so was probably to be attributed to the leukaemia.

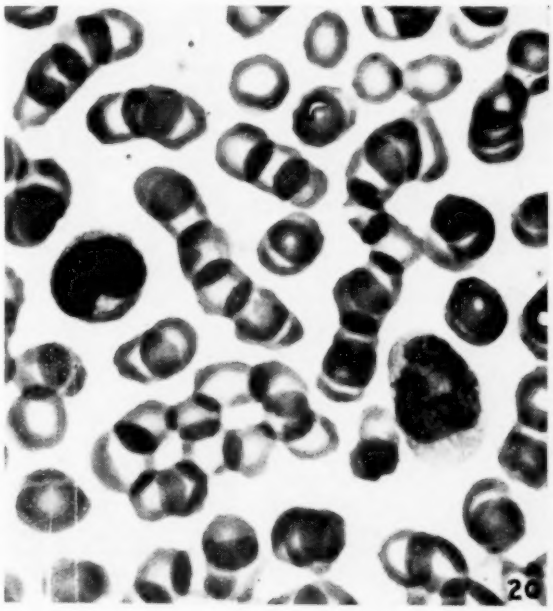


Fig 2. (*c*) Blood Film: Wrights stain (×1200). Further examples of 'monocytoid lymphocytes'.

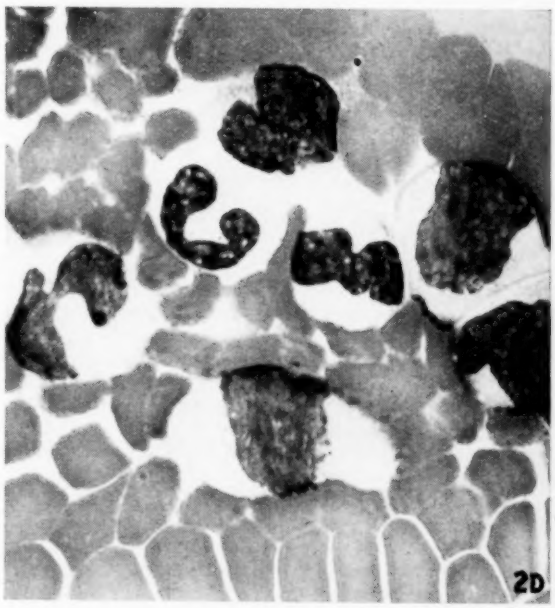


Fig. 2. (*d*) Blood Film: Wrights stain (×1200). Examples of the other type of atypical leucocyte frequently observed which we have reagrded as 'monocytoid myelocytes'.

MATERIAL AND RESULTS

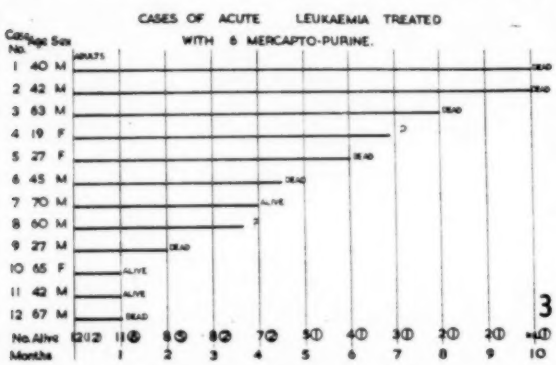
We have had experience of the drug now in a total of 35 cases, and the various cases treated are shown in Table I.

TABLE I. CASES TREATED WITH PURINETHOL

Disease	*No. of Cases*
Acute Leukaemia in Adults	17
Acute Leukaemia in Children	5
Acute 'Blastic' terminal phase of Chronic Myelogenous Leukaemia	7
Chronic Myelogenous Leukaemia ..	1
Reticuloses	4
Chronic Lymphocytic Leukaemia ..	1
	35

Acute Leukaemia in Adults

This is the condition where we have had the most experience in the use of Purinethol and the condition where we believe it has its principal use. It is undoubtedly superior to any other threapeutic agent we possess at present. Purinethol has been used in 17 cases of acute leukaemia in adults. Fig. 3 summarizes our findings in



This figure shows graphically the survival of 12 cases of acute leukaemia in adults treated with Purinethol. The figures show the number alive each month after diagnosis and the figures in parenthesis show the number alive of a control series not treated Purinethol (see text).

12 cases. In order to see if any significant increase in expectation of life had resulted from Purinethol treatment, a comparison was made of the duration of life after diagnosis of the 12 cases of acute leukaemia which had been encountered immediately before the introduction of Purinethol. All these cases occurred between 1951 and 1954, and all except 2 received antibiotics. Data relative to these 'control' cases is shown in Table II and in Fig. 3. It will be seen that by this assessment there is significant prolongation of life coincident with the use of Purinethol. The effect does not seem attributable to the use of antibiotics or blood transfusions, since these were used in the majority of the control cases. On the whole, considerable clinical benefit ensued in 6 of the 12 cases; cessation of bleeding phenomena, clearing up of mouth and throat ulceration in a remarkable fashion, and general improvement in well-being. Objective benefit, such as diminution of lymphadenopathy and

TABLE II. DATA RELATING TO 'CONTROL' CASES

Case	*Age*	*Sex*	*Survival (months)*	*Treatment*
1.	57	F	1½	Antibiotics. Blood transfusions.
2.	17	M	2½	Aminopterin. Blood transfusions.
3.	29	F	5	Aminopterin. Antibiotics. Blood transfusions.
4.	36	M	1	Blood transfusions. Antibiotics.
5.	42	M	2 wks.	Blood transfusions. Antibiotics.
6.	74	F	1 wk.	Radiotherapy.
7.	58	F	14	Blood transfusions. Antibiotics.
8.	35	M	2	Blood transfusions. Cortisone. Antibiotics.
9.	53	F	1	Blood transfusions. Cortisone. Antibiotics.
10.	30	M	1	Blood transfusions. Cortisone. Antibiotics.
11.	46	M	3	Blood transfusions. Cortisone. Antibiotics.
12.	49	F	1	Blood transfusions. Cortisone. Antibiotics.

splenomegaly was very much less constant. Improvement in the blood picture was also very irregular in its occurence. In most cases leucopenia with apparent diminution of primitive cells occured, but was not consistently matched by clinical improvement; *per contra*, in some (a fewer number) clinical improvement occurred without any appreciable change in the leucocyte picture. The reappearance of platelets was found to be more unequivocal evidence of benefit than any changes in the leucocyte picture. As indicated above, the anaemia was but rarely benefited, even in cases which otherwise had a good clinical and, so far as the leucocyte and platelet pictures were concerned, haematological response to the therapy. It did not appear that corticosteroid therapy had any real synergistic effect when given with Purinethol.

Five of these cases were of the monocytic variety—4 acute and 1 of the 'chronic' variety, having survived for 10 years after diagnosis. This type of leukaemia has been notoriously resistant to all forms of therapy, but 3 of the 4 acute cases had good clinical and haematological remissions, the fourth showing a response in the blood picture without any benefit to the clinical state. The chronic case, an elderly man of 73, had as his main complaint persistent painful ulceration of the tongue, gums, and throat. Treatment with Purinethol proved efficacious in clearing up these lesions and permitted the patient to eat normally once more.

Acute Leukaemia in Childhood

Five cases of acute leukaemia in childhood treated with Purinethol have been observed. Remissions occurred of 6, 5, 3, 3, and 2 months respectively, and in all cases it was possible for the child to return home and be treated as out-patient. The remarks above relative to the adult cases are applicable to the childhood cases.

Myeloblastic Terminal Phase of Chronic Myelogenous Leukaemia

We have treated 7 cases in this phase. In all there was a measure of clinical improvement, but in most it was short lived, resistance to Purinethol appearing in 2-6 months. Purinethol, however, is the only agent so far available which has been found to have any effect on this condition.

Miscellaneous Conditions

Purinethol has been used with considerable benefit in a case of chronic myeloid leukaemia which had become resistant to radiation therapy and Myleran. A satisfactory remission lasting 6 months was induced.

In 4 cases of malignant reticulosis, 2 of which were histiocytic medullary reticulosis, 1 a 'myeloid reticulosis' (Israels) and 1 an atypical lymphosarcoma with leukaemic blood changes, no benefit of any kind ensured from the use of Purinethol. Purinethol was similarly without effect on one terminal case of chronic lymphocytic leukaemia.

DISCUSSIONS

The use of Purinethol in the treatment of leukaemia and allied conditions has been reported by Burchenal *et al.*,[3] Burchenal,[4] Fountain,[5] Hayhoe and Whitby,[6] Hayhoe,[7] and Hall *et al.*[8]

Evaluation of the place of Purinethol in the chemotherapy of leukaemia and allied conditions is difficult; in its favour is the fact that it is the only agent we have capable of producing remissions in the acute terminal phase of chronic myelogenous leukaemia and in monoblastic leukaemia; it also produces remission in adults with myeloblastic leukaemia more often than any other agent.

On the other hand, it has clearly no place in the reticuloses generally and, as all the published reports show, it is very inconstant in its action, causing considerable amelioration in one patient and none in the next. Yet it is our impression that in those cases where clinical benefit has occurred the very distressing symptoms of acute leukaemia have been mitigated for the patient and the end, when it came, has been sudden—very sudden and quite unexpected in some cases—and even this we regard as a blessing in this disease. As Thomas Fuller said of the good physician, 'When he can keep life no longer in, he makes a fair and easy passage for it to go out'.

We wish to express our thanks to the many physicians and practitioners who have cooperated in the study of these patients. Thanks are also due to Mr. M. Ulrich of the photographic department of the S.A.I.M.R. for the photomicrographs.

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CEREBRAL PALSY: THE PLACE OF NEUROSURGERY*

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The object of this paper is to examine the potential role of neurosurgical procedures in the prevention, arrest or rehabilitation of and from a group of maladies included under the somewhat vague and unsatisfactory generic term 'cerebral palsy'. In common usage the term embraces the motor and associated disabilities in individuals suffering from the effects of damage to various parts of the brain.

For obvious reasons most time, money and energy have been directed to the management of these disabilities in children. So much has this been the case that the mere use of the term 'cerebral palsy' has almost invariably been confined to discussions on effects arising from disorders of infancy and childhood. In actual fact 'the prolongation of the span of life, with its increase in geriatric diseases, particularly cardiovascular disease, is rapidly causing a much greater incidence of cerebral palsy in adults than in children'.[1]

Etiological Factors and Morbid Entities

There is some immediate practical value in the classification of cases according to etiology. Tables I-IV, compiled from various sources,[1-4] set out the causes of cerebral palsy in infancy and childhood and summarize certain effects and morbid entities. In all the tables those initial lesions or secondary morbid entities which, to greater or lesser extent, lend themselves to surgical removal are marked with an asterisk.

TABLE I. BROAD ETIOLOGICAL CLASSIFICATION

From:	Prenatal Causes	20%
	Natal Causes	70%
	Postnatal Causes	10%

TABLE II. PRENATAL CAUSES

A. HEREDITARY OR GENETIC, STATIC OR PROGRESSIVE

Rudimentary pyramidal tracts—Spastic paraplegia
Defects in basal ganglia—Atonic diplegias, tremors, athetoses, rigidities
Defects in cerebellum and/or its tracts—Ataxia
Metabolic anomalies: phenylpyruvic amentia
Ectodermal or mesodermal defects: tuberous sclerosis,* neurofibromatosis,* Sturge-Weber syndrome * and other vascular abnormalities*
Familial cerebral degenerative diseases

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

B. CONGENITAL NON-HEREDITARY

Erythroblastosis foetalis
Damage to basal nuclei (Kernicterus)
Athetosis and rigidity
Interference with placental circulation
Toxaemia of pregnancy
Maternal asphyxia
Trauma to uterus or foetus
Foetal cerebral haemorrhage, primary or secondary to cerebral softening or/and
Cortical atrophy of various grades or/and
Subdural haematoma(s)

Effects:
Brain cysts and porencephaly *
Regional, lobular or hemispheral atrophy *
Regional, lobular or hemispheral sclerosis *
Microgyria *
Haematoma *
etc.

Maternal infection: neurotropic viruses, syphilis, toxoplasma
Maternal diabetes, liver damage, hyperthyroidism (treatment with thyroid inhibitors)
? Maternal nutritional and vitamin deficiencies
? Maternal gonadal irradiation

TABLE III. NATAL CAUSES

Anoxia: Brain cysts and porencephaly *
Regional, lobular or hemispheral atrophy *
Regional, lobular or hemispheral sclerosis *
Microgyria *
Haematoma *
Meningocerebral cicatrix *
etc.
Mechanical birth injury
Increased fragility of cerebral blood vessels
Bleeding tendency

TABLE IV. POSTNATAL CAUSES

Trauma
Infection: meningitis, encephalitis (e.g. Strümpell-Marie type), brain abscess *
Neoplasm*, often slowly growing
Drugs: lead,* causing anoxia (anaesthetics, barbiturates).
Anoxia from other causes
Arterial occlusions, including acute infantile hemiplegia of obscure origin *
Cerebral haemorrhage and softening following acute infectious diseases *
Venous occlusions

LESIONS AMENABLE TO DIRECT SURGICAL PROCEDURES

The lesions produced by hereditary or genetic effects seldom lend themselves to direct surgical procedures. These are usually only applicable in instances where the anlage gives rise to some recognizable morbid entity at some time remote from birth. Occasionally tuberous sclerosis occurs in an isolated cerebral lesion which can be removed—the writer has had experience of one such case in an adult woman. An isolated neurofibroma can be removed, but more usually intracranial neurofibromas are multiple and often they are further associated with glioma of an optic nerve or meningiomas.[5, 6] These multiple lesions set a limit to the value of surgery. Isolated and relatively small vascular abnormalities lend themselves to removal or coagulation.[7–10] Extensive removal of abnormal vessels and brain is feasible in some cases of the Sturge-Weber syndrome.[11]

Numerous morbid entities arising from congenital, non-hereditary, natal and postnatal causes lend themselves to surgical removal. Most obvious and satisfactory in the prevention of further development of neurological deficit are the removal of haematomas, the proper handling of head injuries, the treatment of brain abscess. In general, the diagnosis of supratentorial brain tumours in extreme youth does not receive the attention it deserves. Such tumours are not infrequently slow-growing.[1] (*Here an illustration was shown of a fibrillary astrocytoma, which was causing frontal adversive seizures and hemiparesis in a 3-year-old child, and was successfully removed.*)

Some of the lesions so far mentioned as amenable to removal, sometimes only in exceptional cases, in themselves constitute a menace to life or can be taken away before they do other irreparable damage. The same does not usually apply to the end-results of a number of causative factors which promote atrophic and sclerotic lesions. Although ablation of these may be feasible it cannot for this reason alone be considered desirable.

The Atrophic and Sclerotic Lesions

This group includes the great majority of the cerebral palsies of infancy or childhood referred to the neurosurgeon. On the basis of radiological studies after air replacement of cerebrospinal fluid, the following gross entities can be recognized:[12]

1. A general dilatation of the ventricular system, sometimes with enlargement of the pericerebral or pericerebellar subarachnoid space.

2. An exaggerated dilatation of part or the whole of one lateral ventricle.

3. Gross cerebral defects, resulting in lobar sclerosis, 'brain cysts' or porencephaly.

Histological studies indicated that the basic alteration common to local microgyria and these grosser signs of brain damage is a characteristic cellular necrosis of the altered cortex.

At the other extreme of this group of atrophies is the local microgyrus.

In clinical practice these morbid entities account for the greater number of *infantile hemiplegias* and *cerebral diplegias*. The bilateral cerebral involvement in the latter at present place them beyond the reach of direct surgical procedures and the attention of neurosurgeons has been concentrated on the first group.

Infantile Hemiplegias

The clinical picture observed in the infantile hemiplegias is slightly variable, but most commonly the child is brought for consultation with a combination of physical and mental defects. There is a spastic hemiparesis, with fixed contractures at wrist and elbow. The affected limbs are usually shorter and generally smaller than their fellows. The skull usually shows an asymmetry. Athetoid movements may occur on the affected side, on which motor weakness and loss of sensory discrimination will be noted. A homonymous field-defect is commonly observed. Mental development varies from bright to very low grade, and behaviour disorders may be most distressing to the parents. To crown all these infirmities, epileptic seizures may occur with great frequency.

The physical disabilities are permanent. The development to adulthood varies, but often there is persistent mental retardation. Epilepsy remains a major disability.

Electro-encephalography indicates a gross dysrhythmia. Ventriculography shows a dilatation of the ventricular system, most often affecting one lateral ventricle in part or *in toto*. There are some cases, however, which do not show the ventricular dilatation and here there is a gliosis of a hemisphere in place of the more usual atrophic, thinned-out, shell covering a lateral ventricle.

(*Two examples were illustrated to show the great variations in the extent of surgical ablations applied to these cases, depending on the extent of the underlying pathology. In the first instance a teenage girl, who had suffered from focal epileptic fits for many years, had a local microgyrus removed with satisfactory result. In the second, a boy of* 19 *years was subjected to hemispherectomy for very frequent major general epileptic attacks, up to* 15 *per day even while on heavy anti-epileptic medication, also with satisfactory result.*)

Various forms of cerebral cortical ablation for epilepsy have been reported in significant numbers, since 1935.[13] The most significant reports have come from Penfield and his associates,[1, 14, 15] who have stressed the epileptogenic role of a zone of apparently normal cortex adjacent to a grossly damaged area.[1, 14, 16]

During the forties Krynauw, in Johannesburg, removed progressively greater areas in atrophic and sclerosed hemispheres in selected cases of 'infantile hemiplegia'.[13, 17] He developed an operation of almost complete hemispherectomy with sparing of the caudate nucleus, internal capsule and thalamus. After a visit to Krynauw's clinic Sir Hugh Cairns introduced the operation to Britain.[18] In 1953 McKissock of London reported on 18 cases of infantile hemiplegia treated by the Krynauw operation.[19] Others in the USA, Spain and Belgium have reported more limited experience with the procedure.[11, 20–23]

In Krynauw's 12 reported cases there was one death and a second case developed an abscess in the basal stump. The others all showed initial improvement in respect of fits and behaviour disturbances,[17] which were considered as epileptic equivalents.[13] In none was the motor disability aggravated by the operation.[17] Perhaps the successors in Krynauw's unit will give us more up-to-date information on follow-up of his cases and tell of their more recent experiences.

In McKissock's 18 reported cases there was one death from infection. The operation eliminated or greatly reduced epilepsy in 16, and removed temper tantrums in 14. Decreased spasticity of affected limbs resulted in greater freedom and facility of movement, but in 2 hemiplegia was made worse—in these local excision would probably have been better advised. Capacity to learn was favourably influenced.[19] In Obrador's 6 reported cases there were 3 deaths, but hemispherectomies were performed after initial failure of more limited resection to alleviate fits.[22]

There were no deaths among the 8 cases treated by hemispherectomy and reported by French *et al.* The results were comparable to those reported by McKissock, but no cases were made worse in respect of motor disturbances.[11]

The Evaluation of Hemispherectomy

It is not yet possible to evaluate the full results of hemispherectomy for atrophic and sclerotic lesions arising in infancy and very early childhood. The encouraging signs are the testimony of reported cases with very satisfactory reduction in the number of epileptic seizures up to 5 years after operation, the knowledge that in properly selected cases there follows no added speech or motor disturbance, and that apart from a rather protracted convalescence the operation does no harm, if complications do not ensue. *The indication for the operation is strictly limited to occurrence of frequent epileptic seizures or equivalents uncontrolled by medication in reasonable doses.*

The release of spasticity may facilitate movement,[19] but it may also by virtue of this make orthopaedic deformities more apparent.[11] For the latter reason neurosurgeons are less inclined to perform the operation in the absence of gross infantile hemiplegia, but here again the yardstick must be the frequency of severe ictal episodes.

The effect on intellect and personality may be favourable, but only if the patient is psychologically adjusted to suitable surroundings. Unfavourable social and family relationships, extreme poverty for which there is no remedy, and initial very low intelligence, should be accepted as contra-indications to the operation. Only latent and already existing mental abilities can blossom after the brake of an irreparably damaged hemisphere has been removed. A near-vegetable mentality is made worse; in its presence the convalescence with its necessary re-education becomes a useless torture to patient and attendants and the motor status becomes worse than ever. Such has been my own experience after being persuaded to operate as a desperate measure.

Regional Cortical Ablations

The older established regional ablations of meningocerebral cicatrices and limited atrophic and sclerotic areas are still indicated more frequently than hemispherectomy. Again, they are indicated only by the incidence of *frequent ictal episodes uncontrolled by reasonable doses of anti-epileptic remedies.*

The failure of these operations is often due to neglect to remove surrounding epileptogenic, normal-appearing brain-tissue. For this reason very large gross lesions are probably better handled by hemispherectomy, but there is already evidence that nicety of judgment is necessary in making this decision, as witnessed by two of McKissock's cases.[19]

In general it is well to apply to cortical ablations in children the rule that they be done only when seizures are focal and coincide with appropriate clinical neurological motor disability.[24]

Cervical Arteriovenous Anastomosis

Attempts at improving the blood supply to brain cells enmeshed in gliotic tissue have been made from time to time, all with singular lack of success. The latest of these methods has been carotid—internal jugular anastomosis in the neck. A large series of cases was reported in 1950, but the results in children with cerebral palsy were disappointing.[25]

Treatment of Dyskinesias

Sir Victor Horsley opened up a new field of surgery when he performed motor cortical ablation for athetosis in 1909.[26] The dyskinesias sometimes afford the outstanding disability in cases of cerebral palsy. Hemispheral damage and paralysis dominate the clinical picture in cases due to direct mechanical trauma from various causes, choreo-athetosis does so in cases due to anoxia.[1]

'The surgical procedures performed for the relief of involuntary movements have been varied and at times bizarre, largely due to the fact that the physiological basis of the phenomena were not understood[27]—they are still only imperfectly understood. A variety of procedures have included: intracortical alcohol injections,[28] cortical ablations of parts of areas 4 and 6[29, 30] of so-called area 45,[31] interruption of extrapyramidal tracts by section in the anterior columns of the spinal cord,[32] surgical damage to the dentate nucleus,[33] section of spinal nerve roots,[34, 35] operations on the caudate nucleus and internal capsule.[36, 37] New modifications of some of these procedures are still being elaborated but none can yet be claimed as beyond the 'experimental' stage. Suffice it to say that surgical relief can be given to some cases of *severe* dyskinesia—that 'neurosurgery has been of occasional benefit in certain cases of severe athetosis or tremors'.[1]

CONCLUSIONS

Various neurosurgical procedures are indicated in a limited number of cases of cerebral palsy of infancy and childhood. It is essential that certain lesions should be diagnosed early and removed, but in the main the role of neurosurgery is confined to the treatment of associated severe epilepsy and less often that of disabling dyskinesias. To obtain salutary effect and not aggravate disability, cases for operation must be carefully selected. For severe epilepsy a range of cortical excisions, from local removal of a microgyrus to hemispherectomy, are available. For dyskinesia operations on cortex, basal ganglia and spinal cord are sometimes suitable.

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WAT IS DIE MEDISYNE-ONKOSTE VAN 'N DISTRIKSGENEESHEER

As gevolg van vertoë deur die Distriksgeneeshere-Groep van die Mediese Vereniging van Suid-Afrika tot die Departement van Volksgesondheid gerig, om beter besoldiging vir sekere dienste, veral wat medisyne-toelaes betref, het die volgende vrae wel ontstaan:

1. Wat is die goedkoopste wat 'n Distriksgeneesheer sy pasiënte kan behandel sonder om doeltreffendheid van behandeling prys te gee?

2. Is die medisyne-toelaes verbonde aan die verskillende Distriksgeneesheerposte voldoende, al dan nie?

Om die eerste vraag te beantwoord is dit nodig om te bepaal wat onder doeltreffende behandeling verstaan word. 'n Vraag waaroor die mees uiteenlopende opinies bestaan en wat afhang van die oogpunt waaruit die geneesheer die beoefening van mediese praktyk beskou. Byvoorbeeld: 'n pasiënt ly aan 'n gewone verkoue met 'n toe, waterende neus, 'n dikkop-gevoel en geen verhoogde temperatuur of positiewe kliniese tekens in die longe nie. Die een geneesheer sal die pasiënt gerusstel oor sy toestand, hom 'n paar A.P.D.-tablette voorskryf en die pasiënt is oor drie dae beter. Dit kos die pasient 15-17/-.

'n Ander geneesheer het miskien 'n bietjie meer sin vir sy eie finansies as vir dié van sy pasiënt, en neem die feit in ag dat hoe groter die bohaai oor die pasiënt hoe meer die pasiënt se ego gestreel word. Indien die pasiënt dan, ten spyte van die bemoeiing van die geneesheer van sy ernstige siekbed herstel, loop hy gevaar om 'n koronêr te ontwikkel as hy sy rekening ontvang, maar bly hy nietemin die geneesheer dankbaar dat hy sy lewe gered het. Doeltreffende behandeling vir die gewone verkoue soos deur laasgenoemde geneesheer toegepas, behels:

1. 'n Langdurige kliniese ondersoek.

2. Die stelling van die pasiënt se toestand as 'n uiters akute infeksie van die lugweë met sekerlik 'n ‚kol op die long'.

3. Die onmiddelike toediening van 'n antibiotiese inspuiting.

4. Die voorskryf van verdere antibiotika per os.

5. Voorskryf van neusdruppels.

6. Voorskryf van een of ander asetielsalisielsuur-preparaat.

7. Voorskryf van vitamine.

8. Moontlik 2 of 3 herhaalbesoeke met of sonder inspuitings.

Uit die oogpunt van die Distriksgeneesheer waar besuiniging in voorskrifte noodsaaklik is, waar die tydfaktor belangrik is, en waar persoonlike prestige by die pasient nie veel tel nie, beskou ek die volgende vereistes as prakties vir 'doeltreffende behandeling'.

1. Die vasstel van die oorsaak van die siekte.

2. Behandeling van daardie oorsaak met die goedkoopste middel wat 'n doeltreffende uitwerking op die siekte-proses het.

Met bogenoemde as grondslag is daar in 'n groot Distriksgeneesheerpraktyk te werk gegaan en boekgehou van die medisynes wat aan elke pasiënt verskaf is.

Alle ‚voorraad'-mengsels word self opgemaak en nie klaar gekoop nie. Die koste is bereken tot die naaste ¼ pennie vir die werklike medisyne in elke voorskrif, volgens die prys wat aptekers vir die galeniese preparate vra. Die koste van bottels, proppe en etikette is ook bygereken.

Die onkoste van spesiale preparate wat van die Departement van Volksgesondheid verhaalbaar is, is buite rekening gelaat.

Die volgende analise van gevalle behels, wat hierdie verslag betref, die maand November 1955, wat 'n baie gemiddelde maand was met geen groot epidemies van enteritis of influenza, longontsteking ens. nie:

Getal gevalle gesien in dorp	510 nie-blankes
Distrik	215 nie-blankes
,,	110 blankes
Totaal:	835

Hierdie syfer sluit geensins roetinegevalle van sifilis of tering ens. in nie, ook nie personeel van die polisiemag of van die twee gevangenisse nie. Dit is slegs gevalle wat met gewone siektes na die spreekkamer vir ondersoek en medisyne kom.

Getal voorskrifte opgemaak en uitgereik in hierdie groep—685.

Die pasiënte kan min of meer in drie groepe gedeel word:

(*a*) Diegene wat hospitalisasie of spesiale behandeling nodig het. Hulle val buite die reeks vir koste-berekening.

(*b*) Die ernstige siekes wat gewone sowel as spesiale middele nodig het; hulle val gedeeltelik in die groep.

(*c*) Die kroniese siekes, lammes en luies. Hulle behels die groep. Hulle is die oorsaak van ontevredenheid en bly self steeds ontevrede. Hulle is die oorsaak ook van die grootste onkoste vir dokter en staat. Hulle eis medisyne vir siektes wat nie bestaan en nie gediagnoseer kan word nie. Hulle word merendeels met 'n troos van 'n paar aspirin-tablette weggestuur.

Soos voorheen gemeld is die empiriese formules in gebruik en hulle werk in die meeste gevalle doeltreffend.

Die verskillende hoofgroepe is voorgeskryf soos in die volgende tabel:

	Getal voorskrifte	*Onkoste per voorskrif*	*Totaal* £ s. d.
Mist. Alb.	76	8d.	2 10 8
Mist. Sodii Salisiel. ..	63	11½d.	3 0 4½
Mist. Pot. Cit.	14	9d.	10 6
Mist. Cretae	46	15d.	2 17 6
Mist. Exp. Stim. ..	31	12d.	1 11 0
Mist. Exp. Sed. ..	26	12d.	1 11 0
Mist. Carmin. ..	5	5d.	2 1
Mist. Pot. Brom. et Tr. Digit.	14	9d.	10 6
Mist. Sulfonamide ..	25	8½d.	17 8½
Mist. Ferri et Ammon. Cit.	42	13d.	2 5 6
Tab. Ac. Asetiel Sal.	55	3½d.	16 0½
Tab. A.P.C.	24	9d.	18 0
Tab. Sulfonamide ..	25	7½d.	15 7½
Tab. Brewers' Yeast ..	40	6½d.	1 1 8
Tab. A.P.D.	38	9d.	1 8 6
Pill Aperient	42	13d.	2 5 6
Ung. Meth. Sal. ..	16	8d.	10 8
Ung. Sulfonamide ..	13	6d.	6 6
Ung. Sulfuris	3	5d.	1 3
Gemengd	87	7d.	2 10 9
Totaal:	685	9·45 Gem.	26 11 4

Hierdie berekening van pryse is slegs vir die materiaal teen kosprys. Daar is geen voorsiening bygereken vir tyd in verband met die aanmaak van voorraadmengsels of die opmaak van verskillende voorskrifte nie.

Die gemiddelde prys van materiaal per voorskrif werk uit op 9·45 pennies.

Die tydfaktor om die mengsels aan te maak en in bottels op te maak, en om etikette met voorskrifte te skryf en aan te plak, is min of meer as volg:

Om die bestanddele vir 'n Winchester (80 onse) uit te meet en te vul neem, teen 'n vinnige spoed bereken, gemiddeld 20 minute. Om tien bottels te vul en te merk, 10 minute. Dit wil sê, 30 minute om 10 bottels op te maak. As die tydberekening volgens die loonskaal van die Departement van Volksgesondheid geskied, naamlik £1 0s. 0d. per uur, beteken dit 1/- per bottel aan arbeid.

Die opmaak van tablette en salf neem gemiddeld 2 minute vir die uittel, in 'n houer sit en die skryf van 'n voorskrif op die houer. Teen dieselfde tarief van £1 0s. 0d. per uur bereken, werk dit uit op elf uur en veertien minute om die 337 voorskrifte op te maak, dit is bereken tot die naaste pond.

'n Minimum medisyne-toelaag in die betrokke geval behoort dan as volg te wees:

Koste van materiaal	£26 11 4
Tyd van opmaak—348 mengsels @ 1/- elk ..	17 8 0
Tyd van opmaak, 337 voorskrifte vir tablette en salf @ 2 minute per voorskrif	11 0 0
	£54 19 4

Dit wil sê as 'n redelike medisyne-toelaag vir die betrokke Distriksgeneesheersdienste betaal word, behoort dit 'n minimum van £54 19s. 4d. te wees.

Die werklike medisyne-toelaag was £12 10s. 0d. per maand, dit wil sê £150 per jaar.

ASSOCIATION OF PHYSICIANS OF SOUTH AFRICA

A meeting of the Southern Transvaal sub-group of the Association of Physicians of South Africa was held at Medical House, Johannesburg on 1 March 1956.

Haemoglobin-C Disease

Dr. Eugene Baskind demonstrated a case of haemoglobin-C disease in a European male aged 28 years, born in the Cape Province, who had presented with an episode of haematuria. On examination the patient had exhibited splenomegaly and doubtful icterus. Blood examination had revealed no anaemia, a leucocytosis of 22,000 per c. mm., and target cells in the blood smears. Dr. Baskind stated that as far as he could ascertain no case of haemoglobin-C disease had previously been reported in South Africa, and this was only the second case ever reported in a White person. Although haematuria had been recorded as occurring in sickle-cell disease and sickle-cell haemoglobin-C disease, it has not been noted in any of the case reports on haemoglobin-C in the literature. A full urinary investigation had disclosed no other cause for the haematuria in this patient. He remarked that the diagnosis should

be considered in cases of splenomegaly, where target cells were fairly numerous in blood smears and where there was clinical evidence of haemolysis.

Dr. S. M. Lewis discussed the haematological findings in the patient. The electrophoretic findings showed that the patient's haemoglobin was composed almost entirely of C type. In addition the blood showed a markedly decreased red-cell fragility even after incubation. He had just begun to investigate other members of the family and had found one brother to have exactly the same haemoglobin as the patient. Although he had not examined the parents' blood, he presumed that the patient was most likely suffering from a homozygous haemoglobin-C disease.

Dr. C. Komins demonstrated the skeletal findings. The skull had a ground-glass appearance and there appeared to be some porous rarefaction of the lower femora and the tubular bones of the hands.

A Case Nephrocalcinosis

Dr. S. J. Fleishman showed a case of a 42-year-old woman who in 1949 during a routine radiological investigation for abdominal pain had been found to be suffering from nephrocalcinosis. In January 1954 he had seen this patient because of profound weakness—she was able neither to sit up in bed nor lift her head from her pillow. Her symptoms included polyuria and polydypsia which had commenced at the time of abdominal pain. Examination had revealed an acidotic type of breathing and very brisk reflexes. Biochemical investigations had shown a marked hypokalaemia (2·3 mEq/l) as well as acidosis (CO^2 combining power 6·7 mEq/l), hyperchloraemia (120 mEq/l) and a blood urea of 72 mg.%. She was diagnosed as nephrocalcinosis, renal acidosis and potassium losing nephritis, and rapid improvement in the symptoms ensued after massive doses of potassium citrate. Recently further biochemical studies had been made of the sweat and saliva. The potassium and sodium content of these secretions had been measured. Although the investigations were not fully completed it appeared from these results that a diagnosis of primary aldosteronism was not altogether unlikely, despite the finding of acidosis.

The radiological appearance of the nephrocalcinosis was demonstrated by Dr. E. Price. He discussed the causes and drew attention to the fact that renal biopsy in Conn's case had shown the presence of microscopic calcification, and the finding of nephrocalcinosis was therefore not necessarily incompatible with the diagnosis of an aldosterose tumour of the adrenal.

Dr. B. Senior pointed out that the sodium and potassium figures for both the saliva and sweat were markedly outside the normal range. Experience was limited but comparison with published figures indicated excessive loss of potassium and retention of sodium; the low sodium excretion was particularly significant as the flow rates of the sweat and saliva were high and in normal subjects there is increased sodium loss with increase of flow. The findings in this patient suggested a dynamic process causing conservation of sodium and loss of potassium and this again raised the possibility of hyperaldosteronism. The published reports of this entity however differed in one particular regard, namely a tendency towards alkalosis, whereas the feature in this patient was acidosis. As in states of potassium-depletion there may be an impairment of renal function reflected by an inability to pass a concentrated urine, he suggested that in some cases further renal dysfunction might occur resulting in defective acidification of the urine with consequent systemic acidosis.

The implications of the possible relationship between renal acidosis and hyperaldosteronism were then discussed.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. Louis Blumberg, M.B., Ch.B (Cape Town) F.R.C.S. (Eng.) has commenced practice as a specialist surgeon at 501 Commercial Union Buildings, St. George's Street, Cape Town. His telephones are: Rooms 2-8800, Residence 7-9548.

* * *

Special Bursaries for Research in Germany. The *Deutscher Akademischer Austauschdienst* is offering special bursaries for qualified research workers who are desirous of cooperating with German scientific institutions for a period of 3-6 months during the academic year 1956/57. They amount to DM 300-350 (£25-£30) per month. Applications are invited from South African research workers on forms which are obtainable from The Press and Cultural Affairs Attaché, Embassy of the Federal Republic of Germany, P.O. Box 2023, Pretoria (Telephone 3-5291). Applications should reach the office of the Secretary, Department of Education, Arts and Science, Van der Stel Buildings, Pretoria, not later than 15 April 1956.

* * *

Congres International Pour Le Latin Vivant. The First International Congress to make Latin a Living Language will be held in Avignon, France, on 2-6 September 1956, under the auspices of the Ministry of International Education, the University of Aix-en-Provence, the Alliance Francaise and the City of Avignon. The aim of this congress is to determine the role Latin should play in modern times and to study practical methods for making it a living language.

MEDICAL AND GOODWILL TOUR OF EUROPE

A few vacancies remain for the Medical and Goodwill tour of Europe arranged for doctors and their families lasting from Sunday 27 May until Monday 13 August. The tour will give those wishing to take part in it an opportunity to visit Italy, France, Holland, the United Kingdom, Switzerland, Germany, Sweden, Norway and the Fjords, and Denmark. Special extension to Israel, the U.S.A. or anywhere else can be arranged.

Full particulars of the tour may be had from Dr. J. H. Struthers, President, South African Medical Association, General Hospital, Pretoria, or from Dr. E. B. Woolf, M.P.C., 34 Jan Smuts Avenue, Forestown, Johannesburg.

Those taking part will leave Johannesburg by Sabena plane on 27 May and the tour will take in Rome, Florence, Venice, Milan, Rapallo, San Reimo, Nice, Zurich, London, Brighton, Hamburg, Stockholm, Narvick, Tromso, Bergen, Oslo, Gothenburg, Copenhagen, Amsterdam and Paris.

Complete arrangements have been made for visits to places of historic and cultural interest and travel and hotel accommodation has been arranged.

BOOKS RECEIVED RECENTLY AT THE WITWATERSRAND MEDICAL LIBRARY

Beckett, R. H. Modern actinotherapy. London. Heinemann. 1955.

Beier, A. O. Wildwachsende Heilpflanzen und Teekräuter. Hannover. Trowitsch. 1948.

Bonica, J. J. The management of pain. London. Kimpton. 1953.

Brain, W. R. Diseases of the nervous system. 5.ed. London. O.U.P. 1955.

British Medical Association. The recognition of intoxication. London. British Medical Association. 1954.

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Büchner, F. Handbuch der allgemeinen Pathologie. Berlin. Springer. 1955. Library has v. 2. Das Cytoplasma.

Cantarow, A. Clinical biochemistry. Philadelphia. Saunders. 5.ed. 1955.

Charlewood, G. P. Bantu gynaecology. Johannesburg. Photo Publ. Co. of S.A. 1956.

Ciba Foundation. Ciba Foundation symposium or porphyrin, biosynthesis and metabolism. London. Churchill. 1955.
Cowdry, E. V. Cancer cells. Philadelphia. Saunders. 1955.
Dale, H. H. An autumn gleaning. London. Pergamon press. 1954.
Dellinger, G. Herbarium des Georg Dellinger anno 1553 zu Nürnberg. Salzburg. Akademischer Gemeinschaftsverlag.
Drinker, C. K. The clinical physiology of the lungs. Springfield. Thomas. 1954.
Fischer, R. A. Design of experiments. 6.ed. Edinburgh. Oliver & Boyd. 1951 reprinted 1955.
Fuchs, L. Alt-Kräuterbüchlein von der Kraft und Wirkung der Kräuter. Heilbronn. Salzer. 1951.
Gibbens, J. The care of young babies. 4.ed. London. Churchill. 1955.
Gillilan, L. A. Clinical aspects of the autonomic nervous system. London. Churchill. 1955.
Goulden, W. O. German-English medical dictionary. London. Churchill. 1955.
Greisheimer, E. M. Physiology and anatomy. 7.ed. London. Pitman. 1955.
Hertwig, H. Gesund durch Heilpflanzen. Berlin. Siebeneicher Verlag. 1950.
Hunter, D. The diseases of occupations. London. English U.P. 1955.
Ilingworth, C. F. W. A short textbook of surgery. 6.ed. London. Churchill. 1955.
Kimber, D. C. Textbook of anatomy and physiology. 13.ed. New York. Macmillan. 1955.
Künzle, J. Das grozze Kräuterheilbuch. 12. Aufl. Olten. Walter. 1945.
Kugelmass, I. N. The management of mental deficiency in children, New York. Grune & Stratton. 1954.
Lamb, J. P. Commercial and technical libraries. London. Allen & Unwin. 1955.
Leyel, C. F. Herbal delights. London. Faber. 1937.
McIlwain, H. Biochemistry and the central nervous system. London. Churchill. 1955.
Makar, N. Urological aspects of bilharziasis in Egypt. Cairo. Societé orientale de publicité. 1955.
Mellors, R. C. Analytical cytology. New York. Blakiston.
National Society for Crippled Children and Adults. A manual of cerebral palsy equipment. Chicago. National Society for Crippled Children. 1950.
Nuffield Provincial Hospitals Trust. Report on an experiment in hospital costing. Oxford U.P. 1952.
Onians, R. B. Origins of European thought about the body, the mind, the soul, the world, time and fate. 2.ed. Cambridge U.P. 1954.
Paterson, D. Sick children. 7.ed. London. Cassell. 1956.
Quelch, M. T. Herbal remedies and recipes and some others. London. Faber. 1945.
Rawling, L. B. Landmarks and surface markings of the human body. 9.ed. 1953. London. Lewis.
Reddish, G. F. Antiseptics, disinfectants, fungicides and chemical and physical sterilization. London. Kimpton. 1954.
Riddell, P. I was an alcoholic. London. Gollancz. 1955.
Savage, B. Preliminary electricity for the physiotherapist. London. Faber. 1954.
Schauffler, G. C. Pediatric gynecology. 3.ed. Chicago. Yearbook publishers. 1953.
Storey, G. M. Thoracic surgery for physiotherapists. London. Faber. 1955.
Taylor, A. S. Principles and practice of medical jurisprudence. 11ed. London. Churchill. 1956. v. 1.
Theodoric. The surgery of Theodoric. New York. Appleton-Century. 1955.
Thorpe, W. V. Biochemistry for medical students. 6.ed. London. Churchill. 1955.
Tidy, N. M. Massage and remedial exercises. 9.ed. Bristol. Wright. 1952.
Weinmann, J. P. Bone and bones. 2.ed. London. Kimpton. 1955.
World Health Organization. Control of insect vectors in international air traffic. Geneva. W.H.O. 1955.

BOOK REVIEWS : BOEKRESENSIES

PREVENTIVE MEDICAL PRACTICE

The Prevention of Disease in Everyday Practice. By Isadore Givner, B.S., M.D., F.A.C.S. and Maurice Bruger, M.Sc., M.D., C.M., F.A.C.P. Pp. 964 with illustrations. £8 10s. 0d. St. Louis: The C. V. Mosby Company. 1955.

Contents: 1. A Practical Outline of Cancer Prevention. 2. Prevention of Poisoning—Accidental and Intentional. 3. Preventive Pediatrics. 4. Preventive Aspects of Infectious Diseases. 5. Prevention of Deformities in the Orthopedics of Childhood. 6. Prevention of Deformities in the Orthopedics of Childhood. 7. Prevention of Deformities Following Acute Anterior Poliomyelitis. 8. Preventive Hematology. 9. Preventive Aspects of Diabetes Mellitus and Spontaneous Hypoglycemia. 10. Preventive Cardiology. 11. Preventive Aspects of Peripheral Vascular Disease. 12. Preventive Aspects of Pulmonary Disease. 13. Preventive Aspects of Gastroenterology. 14. Preventive Aspects of Diseases of the Liver, Bile Ducts, and Pancreas. 15. Preventive Aspects of Arthritis and Allied Rheumatic Disorders. 16. Preventive Aspects of Tropical Medicine. 17. Preventive Aspects of Allergic Disorders. 18. Preventive Dermatology. 19. Preventive Psychiatry. 20. Preventive Neurology. 21. Preventive Radiology. 22. Preventive Aspects of Industrial Medicine. 23. Preventive Aspects of Surgery. 24. Preventive Anesthesiology. 25. Preventive Aspects of Abdominal Surgery. 26. Preventive Aspects in the Treatment of Fractures and Other Injuries. 27. Preventive Aspects of Thyroid Surgery. 28. Preventive Aspects of Thoracic Surgery. 29. Preventive Aspects of Cardiac Surgery. 30. Preventive Ophthalmology. 31. Preventive Otolaryngology. 32. Preventive Laryngology and Bronchoesophagology. 33. Preventive Aspects of Neurological Surgery. 34. Preventive Proctology. 35. Preventive Urology. 36. Preventive Aspects of Obstetrics. 37. Preventive Gynecology. 38. Preventive Dentistry.

This book consists of 41 monographs written by 47 practitioners, each of whom is a specialist in his subject. The book is not intended to be read like an ordinary text-book for is is really a reference book and covers almost the whole of medicine and surgery. It sets out to provide the general practitioner with reasoned guidance in the *prevention* of a large number of diseases and allied conditions and it is written in clear style, somewhat verbose in parts.

To achieve their object the writers have had to consult an enormous volume of literature, most of which is American; and the nature of their task made it imperative that all conditions should be considered from more than just the preventive angle. This is both understandable and desirable, since prevention can be based only on a many-sided knowledge of the disease entities which it is the intention to prevent. All this has tended to swell the volume of this book without in the least appearing to be redundant, or out of place.

As each monograph stands on its own, it would be very difficult to select any as of outstanding value compared to others without being unfair to the individual authors. Yet this reviewer could not help enjoying the monographs on Cancer, Paediatrics, Diabetes and Tropical Medicine, especially the last-named.

It is interesting to read that in the matter of cancer prevention the Vitamin-B complex as well as iron seem to enhance the resistance of the tissues to cancer in certain situations such as the mouth, pharynx and oesophagus, and that an abuse of tobacco and alcohol may deprive the tissues of these protective accessory foods. Elsewhere 'universal circumcision' is advocated as a preventive measure of practical value against cancer of the cervix, penis and prostate. Wherever good penile hygiene, especially circumcision, is practised cancer at these sites is lowest, whereas the incidence increases 3-fold where it is rare.

All advice throughout the book seems based on well founded observation and common sense. Still one would rather not *tell* a pregnant woman that she is Rh-negative while her husband is Rh-positive and that trouble may be in store for them; there may be nothing after all. Such telling will not *prevent* trouble for the perhaps anxious mother.

One author states that 90% and more of people today appear to suffer from what he calls 'spontaneous hypoglycaemia' with symptoms ascribable to anxiety and stress in our everyday life. It is to be regarded as a 'stimulative hypoglycaemia' and chiefly functional in nature and its effects are removed by high protein and low carbohydrate diet; 'a difference of opinion exists, however, as to the optimum number of meals or feedings which should be prescribed per day'.

Since many so-called tropical conditions and diseases also

present themselves in other regions (this reviewer has recently seen one case of flagrant pellagra mixed with beri-beri in the OFS), one author is of the opinion that prevention of such conditions should be brought to the notice of practitioners working outside the tropics as well. Such knowledge will tend to dissipate exaggerated and distorted ideas about tropical conditions resting on unscientific basis.

The book is crowded with most useful information emphasizing the fact that we have progressed far on the way towards 'warding off sickness and death' in a 'collective' manner although much has still to be learnt about many disease conditions requiring preventive measures based on such knowledge. This book is therefore an excellent attempt in this direction. Future editions should prove even more useful to practitioners in many branches of medicine. The book itself is well printed and well bound.

G. C. A. v. d. W.

UROLOGICAL PRACTICE

Urological Practice. Roger W. Barnes, B.A., M.S., M.D., F.A.C.S., F.I.C.S. and Henry L. Hadley, B.A., M.D., D.N.B. (Pp. 494 with 166 illustrations. £5 6s. 3d.) St. Louis. The C. V. Mosby Company. 1954.

Contents: Part I. Section 1. Leading Symptoms, Signs and Findings in Urogenital Diseases—An Index of Urogenital Symptoms Outlined for Use in Diagnosis. *Section II.* Symptomatic Treatment of Common Urogenital Complaints—An Index of Urogenital Complaints outlining the Symptomatic Treatment of Each. *Part II.* A Discussion, for the General Practitioner, of Genitourinary Diseases. 1. Genitourinary Diagnostic Procedures. 2. Differential Diagnosis of Abdominal Pain in Relation to the Diagnosis of Acute Abdominal Conditions. 3. Urogenital Infections. 4. Urinary Calculi (Urolithiasis, Urinary Stones). 5. Diseases of the Penis. 6. Malformations of the Penis and Urethra. 7. Diseases and Injuries of the Anterior Urethra. 8. Lesions of the Scrotum and Its Contents. 9. Surgical Procedures Applied to the Scrotum and Its Contents. 10. Infertility in Men. 11. Infertility in Women. 12. Sexual Maladjustment. 13. Chronic Congestion (Toxic Hypertrophy) of the Prostate. 14. Infections of the Posterior Urethra, Prostate and Seminal Vesicles. 15. Benign Prostatic Hyperthrophy. 16. Carcinoma of the Prostate. 17. Miscellaneous Diseases, Injuries and Malformations of the Prostate, Posterior Urethra and Seminal Vesicles. 18. Surgery of the Prostate. 19. Urethral and Bladder Disorders in Women. 20. Cystitis and Infections of the Bladder. 21. Vesical Calculi (Bladder Stones). 22. Tumours of the Bladder (Vesical Neoplasms). 23. Miscellaneous Diseases of the Bladder. 24. Surgical Procedures Applied to the Urinary Bladder. 25. Enuresis (Bed-Wetting). 26. Diseases, Injuries and Malformations of the Ureter. 27. Hydronephrosis (Pyelolaciectasis). 28. Renal and Perirenal Infections. 29. Nonsurgical Diseases of the Kidney. 30. Movable Kidney. (Nephroptosis). 31. Kidney Stones (Nephrolithiasis, Renal Calculi). 32. Tumours and Cysts of the Kidney and Perirenal Space. 32. Malformations (Anomalies) of the Kidney. 33. Injuries and Surgery of the Kidney.

As will be noted from the contents, the first part of this book consists of an alphabetically arranged list of leading urological symptoms and signs. This is followed by an index of common urological complaints with the symptomatic treatment of each. This section is presumably included for easy and quick reference but, because of its brevity, is of doubtful value.

The second and main part of the book consists of a discussion of genito-urinary diseases for the general practitioner. No major surgical techniques are included, while stress is laid on office procedures. This section is very comprehensive and the subject is well covered. In a brief review of this nature, it is impossible to comment on individual chapters. A few remarks made at random follow: An unusual method of circumcision is presented and it is surprising to the reviewer that circumcision is suggested as the immediate treatment for paraphimosis. The section dealing with the technique of passing urethral instruments is well written. The reviewer notes that the authors suggest the use of sclerosing solutions in the treatment of hydroceles, without stressing the hazards. The rapid evacuation of an overdistended bladder is now accepted as perfectly safe, provided all precautions to prevent sepsis supervening, are taken.

The illustrations and radiographic reproductions are excellent. The book is unfortunately expensive and because of its limited field, is unlikely to suit the needs of the average general practitioner.

G.D.

ENDOCRINOLOGY

Textbook of Endocrinology. Second Edition. Edited by Robert H. Williams, M.D. Pp. 776+xii with 173 illustrations. £5 10s. 6d. Philadelphia & London: W.B. Saunders Company 1955.

Contents: 1. General Principles of the Physiology of the Endocrines. 2. The Pituitary. 3. The Thyroid. 4. The Adrenals. 5. The Testes. 6. The Ovaries. 7. The Pancreas. 8. Diseases of the Parathyroid Glands. 9. The Influence of the Endocrine Glands Upon Growth and Development. 10. Neuroendocrinology. 11. Obesity. 12. Laboratory Diagnostic and Assay Procedures. 13. Diagnosis and Treatment of Endocrinopathies; Hormone Preparations. Index.

In the 5 years that have elapsed since the 1st edition was published, the field of endocrinology has expanded so considerably that it has been necessary to re-write almost every chapter. In most instances the articles are remarkably up-to-date, and include references to many of the advances which were published during 1955. Thus one finds comments on such recently reported substances as triiodothyronine and aldosterone.

The contributors include such well-known experts as Lawson Wilkins, Reifenstein and George Thorn. The editor has himself contributed several chapters and his dissertation on the thryroid is of an exceptionally high standard. Unfortunately, some of the other articles are less worthy of praise. The discussions on sexual precocity and on the diagnosis of the different varieties of intersexes are distinctly poor. Careless editing is no doubt responsible for the reference to the XX chromosomes of the male!

The editor rightly stresses that the endocrine glands are not responsible for such conditions as obesity, mental retardation and homosexuality, and he strongly deprecates the indiscriminate usage of hormones, e.g. thyroid, in the treatment of these and other non-endocrinal conditions.

It is a pity that the volume possesses so few illustrations. Endocrinology, perhaps more than any other branch of medicine, demands an awareness of the appearances in the various disease states and this text-book would benefit by the inclusion of many more reproductions.

Finally, one must criticize the relative lack of recognition of work done outside of America. For instance the outstanding contributions of Dent and other British investigators in the field of calcium metabolism are not mentioned at all.

The accent in this volume lies on physiological considerations rather than clinical. This will make the book particularly valuable to those wishing to acquire an understanding of fundamental endocrinology. The established worker in the field will also benefit from a perusal of its contents.

R.H.

DIAGNOSTIC CLASSIFICATION OF TUBERCULOSIS

A New Classification of Tuberculosis with New Diagnostic Standards. By Milosh Sekulich, M.D. Pp. 63. 3s. 6d. London: William Heinemann Medical Books Ltd. 1955.

Contents: 1. History of the New Classification. 2. The Classification and Diagnosis. 3. Notation and Terms Used in the Classification. 4. Definitions of Types, Forms and Sub-Forms. 5. Differentiation of Primary from Secondary Tuberculosis. 6. Classification of Non-Pulmonary Tuberculosis. 7. How to Run a Chest Clinic. 8. Crucial Clinical Evidence in Favour of the Classification. 9. A Minimum Basic Classification for Epidemiological Purposes. Index.

In a brochure of 63 pages, the author discusses a classification, the result of many years' personal clinical observation. He classifies pulmonary tuberculosis thus:

1. *Primary Type*
    1. Inflammatory form (benign primary).
    2. Caseous form (Malignant primary).
2. *Secondary Type*
    1. Fibro-caseous form.
    2. Fibrous form.

He divides these forms into 29 sub-forms. The extent of the lesion is described by zones in the usual way. Eight different degrees of activity are suggested.

In the classification of non-pulmonary tuberculosis 23 terms are used.

A claim is made that the application of this classification can 'render the work of chest clinics at least 100% more efficient and economical than they are to-day'. One wonders, however, if the use of such terms as 'invisible benign primary tuberculosis' or 'quiescent malignant primary tuberculosis' is really helpful in the efficient administration of a tuberculosis service.

B.A.D.

MODERN PSYCHOSOMATIC MEDICINE

Modern Trends in Psychosomatic Medicine. Edited by Desmond O'Neill. Pp. 375 + xi with 29 illustrations. London: Butterworth & Co. (Publishers) Ltd. 1955.

Contents: 1. The General Practitioner and the Psychosomatic Approach. 2. The Psychosomatic Concept in Medicine. 3. Doctor, Patient and Student. 4. The Psychosomatic Approach in Paediatrics. 5. Significance of the Family Setting in the Evolution of Infantile Acrodynia. 6. Limb Pains in Children. 7. Psychological and Social Aspects of Sydenham's Chorea. 8. Studies on Ulcerative Colitis: Personality Structure, Emotional Conflict Situations and Effects of Psychotherapy. 9. A Psychiatric View of Skin Disorder. 10 Suggestion and Hypnosis in Obstetrics. 11. Constitutional Aspects of Psychosomtaic Medicine. 12. Essential Hypertension. 13. Thyrotoxicosis. 14. Emotion and Eye Symptoms. 15. Psychogenesis and Psychotherapy of Bronchial Asthma. 16. Sexual Adjustment and Bodily Illness. 17. Anxiety and Muscle Tension. 18. Music and Migraine. 19. Group Psychotherapy in Psychosomatic Disorders. 20. Analytic Therapy. 21. Abreaction Therapy of Psychosomatic Disorders. Bibliography.

This book represents a phase of considerable significance in the development of medicine. It indicates the extent to which the psychological aspects of illness, for too long repressed, are now being re-integrated in the practice of medicine.

It is exceptionally well planned and the editor deserves our grateful thanks for the way in which he introduces these modern trends in psychosomatic medicine. The earlier contributions include little of the so-called 'jargon' to which so many practitioners take exception in other psychiatrically oriented studies. It is by a slow process that we are brought to the later sections of the symposium in which technical terms are more frequently used. It is indeed a tribute to the insight of the editor in his job of educating the profession to those skills which are so needed in practice.

It is not possible to single out for discussion any particular chapter of the 21 stimulating essays. The introductory chapter by a general practitioner describing his own growth towards psychosomatic thinking and of the satisfaction he has from the translation of this thinking into his practice strikes the keynote of sensitivity to patient needs which characterizes the book. To the physician in his ward and the teacher with his student this book is a vital contribution, but to the family physician it is indispensable—a must which is both enjoyable and stimulating. Apart from its content, the publishers have made it one of those books we like to handle and read.

S.L.K.

BIOCHEMISTRY AND THE CENTRAL NERVOUS SYSTEM

Biochemistry and the Central Nervous System. By Henry McIlwain, Ph.D., D.Sc. Pp. 272+vii with 43 illustrations. 40s. 0d. London: J. & A. Churchill, Ltd. 1955.

Contents: 1. Biochemical Studies of the Brain. 2. Metabolism of the Brain *in situ*. 3. The Chemical Composition of the Brain. 4. Metabolism of Separated Cerebral Tissues. 5. Cell-Free Cerebral Systems; Glycolysis and an Oxidative Pathway. 6. Pyruvate Metabolism; Oxidative Phosphorylation. 7. Amino-Acids and Cerebral Activities. 8. Vitamins and the Central Nervous System. 9. Cerebral Lipids. 10. Cytochemical and Histochemical Aspects. 11. Chemical and Enzymic Make-Up of the Brain During Development. 12. Acetylcholine, Sympathin and Related Substances. 13. Depressants and Excitants of the Central Nervous System. 14. The Speed of Chemical Change in the Brain. Author Index. Subject Index.

This book is based on lectures given in the courses of physiology and psychological medicine in the University of London. It gives a clear account of the progress which has been made in our knowledge of the chemical constitution of nerve tissue and the chemical changes which underlie the activity of nerve cells.

Many of us have rather a dim and distant acquaintance with the chemical physiology of the central nervous system; we may have gathered that the brain has a blood supply which appears lavish for the work it does, that it needs an uninterrupted supply of oxygen and carbohydrate, and that glutamic acid is a mysterious nerve food for backward children and may be of help in hepatic coma.

The details of these and the many other facets of cerebral metabolism are described here as far as they are known at present: these details will be very valuable to a select band of physiologists and other advanced workers. But others, less exalted, may carry away general conceptions of great interest and importance; amongst these will be a realization of the enormous size of the cerebral respiratory exchanges and of the large number of chemical processes now known to be concerned; but perhaps most striking of all is the speed with which chemical changes take place so that a delay of a second or less may cause important chemical transformations to be missed altogether. In spite of such difficulties and the complexity of some of the substances concerned the progress recorded is very gratifying.

G.C.L.

CORRESPONDENCE : BRIEWERUBRIEK

TWO CASES TREATED BY HYPNOSIS

To the Editor: May I record a further note about a patient mentioned in my letter[2] published under this heading on 7 April, 1956.

The patient Miss M.B. returned a month later for her second dental appointment. As before, we first had a session in my surgery; but on this occasion I was unable to accompany her to the dentist's rooms and actually induce the hypnosis there. I suggested to her under hypnosis that she would respond to the dentist's instructions in exactly the same way as to mine.

My sentence for the induction of the hypnosis was, 'I want you now to go into a deep sleep.' To wake the patient—'You can now wake up'. I did not induce any amnesia during this preparatory session and allowed the patient to participate completely in this proposed transference. I then wrote a note to the dentist describing our preparations and giving him the two key sentences.'

He telephoned to me an hour later, and said he had had to repeat the induction command. The patient afterwards told me, 'The first time he omitted the word "now", and nothing seemed to happen. The second time he read the sentence straight from your note, and I felt my eyes grow heavy and close.' Complete analgesia had been produced and a major filling completed.

On this occasion the patient felt and heard the drill in her mouth. She again saw scenes from the film, 'The Robe', but stated that this time she did not seem to enjoy it ... Despite the fact that no direct suggestions were given that she would see the film again, the similarity between this session and her previous session was so strong that she herself seems to have produced the hallucination of the film scenes.

It should perhaps be pointed out that this patient is not unique. Any person in whom a trance state can be induced can experience these phenomena. According to Hull[2] a trance state can be induced in 34% of patients.

B. W. Levinson

S.A.W.A.S. Hall
Main Road
Muizenberg
3 April 1956

1. Levinson, B. W. (1956): S. Afr. Med. J., **30**, (7 April).
2. Hull, C. L. (1933): *Hypnosis and Suggestibility.* New York: Appleton—Century—Crofts.

POLIOMYELITIS ANTIBODY TESTS

To the Editor: We have has so many requests for information regarding the tests for immunity against poliomyelitis that we should be grateful for an opportunity to explain the value of these tests and the precautions to be taken in collecting the blood for them.

The test is based on the finding that serum with antibody protects tissue cultures from the destructive effect of the corresponding poliovirus. It is thus possible to determine the presence or absence of each of the three types of poliovirus antibody respectively.

For this test about 5 c.c. of blood collected in a clean sterile syringe free from antiseptics is necessary. This blood should be sent in a sterile tube to The Poliomyelitis Research Foundation, c/o the South African Institute for Medical Research, P.O. Box 1038, Johannesburg. The test takes up to 7 days to complete and may take longer if it is necessary to repeat it for any reason, as often happens. The results of the test indicate whether an individual has antibody against each of the three types of poliovirus.

It has been shown that one or other type of poliovirus antibody does not protect against infection with the remaining types of poliovirus. The results of the test are therefore of value in assessing the need for vaccination against poliomyelitis. Children having antibodies against all three types of poliovirus do not need to be vaccinated, but the need for vaccination of children lacking one or other of these antibodies should be considered.

J. H. S. Gear
Director of Research

Laboratories of the Poliomyelitis Research Foundation
P.O. Box 1038
Johannesburg
28 March 1956

THE SPECIALIST REGISTER

To the Editor: Much is still being written about the specialist register, but I think that it is still insufficiently realized that the specialist register not merely encourages but forces the profession to break one of its most important ethical rules, the rule of supersession—a rule designed to protect the public as well as to enhance medical practice and to promote the dignity of the profession.

In South Africa the public has been and is being educated to select specialists themselves under the misleading slogan of 'free choice of doctor'; in Great Britain they are being taught that to do so is not in their own interests. I quote from *Family Doctor* (February 1956), an excellent and bright journal for the lay public, issued by the British Medical Association, and edited by Dr. I. H. Flack:

'We get lots of letters from readers about specialists, good letters from good people with interesting problems. And over and over again they say, "Please could you recommend a specialist."

'Now *Family Doctor* can do most things for you, but this is one of the the things we can't do. Let's say you've a headache and you feel you need a specialist. It could be due to a defect of vision and you might be greatly helped by an ophthalmologist. It might be your sinuses and then the chap to help is an oto-rhino-laryngologist, who is an ear, nose and throat doctor off duty.

'It could be high blood pressure and you may need a consultant physician to get it really sorted out. It could be something inside your skull that only a neuro-surgeon could cope with. It could be.... It could be any one of forty-nine different things all calling for different remedies by different specialists.

'It's the same with most symptoms. There are many possible causes. Therapy is but quackery unless it is designed to tackle the cause of what ails you. What you need, in fact, is not a specialist but your own general practitioner.

'He or she will sort out for you all the possible causes and arrive at a diagnosis which makes treatment sensible and rational. If you do need a specialist, he will tell you so and make the necessary arrangements and appointments.

'Choosing your own specialist is a bit like playing Russian roulette with one round in a six-chambered revolver. It might work out fine—or it might not.'

Free choice of doctor is a motto designed to emphasize our opposition to closed panels; it cannot ligitimately be used in a question of another kind altogether—the patient/doctor/specialist relationship.

Sen. G.P.

26 March 1956

THE GENERAL PRACTITIONER AND THE CONSULTANT ON THE HOSPITAL STAFF

To the Editor: Mr. McMurray's fair and factual article on *General Practitioners and Hospitals*[1] recalls to me a memorandum I wrote in 1948 suggesting a clinical partnership between the general practitioners and the consultants on the staff of a certain hospital, and indicating a method of approach.

The honorary medical officer (a general practitioner) occupies a key position in this hospital. As in other provincial hospitals he has absolute control over his cases; he investigates and treats them as he thinks necessary and calls a consultant when he considers it advisable. He, and he alone, decides how often to visit the children's wards, how often to examine the patients, when to investigate the details of their dietetic management, etc. He also is the sole judge at what stage during the child's sojourn in hospital he needs the consultant's aid. This is as it should be; the medical officer is the captain on the bridge. The hospital board has appointed consultants—but they can only be used at the discretion of the honorary medical officer.

The consultant's duty is clearly to come to the aid of the child at the request of the medical officer. In hospital the consultant can dispense with the actual presence of the medical officer, because the resident and sister are there and all the relevant clinical data, charts, blood counts, X-rays, etc. But the consultant is no longer the magician of old, and the notion of his running a neck-and-neck race with the Angel of Death has long passed. He expects to be treated as a colleague and given an opportunity of doing something effective. He is as human and has the same limitations as the medical officer in charge; and is only in a better position because he is 'the last man'. In order to be useful he demands certain essentials; *otherwise his services are frustrated and the patients suffer* He needs the following:

(*a*) A good history. This is essential with child patients who cannot tell you themselves. The medical officer in charge should get the history from the parents, or instruct the resident to do so (it is the resident's primary duty).

(*b*) A short discussion with the medical officer in charge. In this way the consultant can at least know what the medical officer has observed.

(*c*) An opportunity to do something effective. This is only obtained when the consultant is called in *early*—not in the last stages when the child has been in the hospital for weeks.

In the children's wards a unique type of case is admitted—very unlike most cases seen in private practice. Some of the children are in a dreadful physical state. They demand a great deal of scientific investigation and personal supervision over the minutiae of treatment; a daily estimate of the clinical condition, full reports on urine, blood and faeces, and meticulous care in the dietary regimen. The consultant's position is difficult; he is not like the surgeon, who takes over the case and treats it himself. *One* consultation is totally inadequate, and yet he is expected to accept responsibility, and he does accept it.

The experienced practitioner knows that these cases cannot be treated in a single consultation. The paediatrician is placed in much the same position as a physician would be if he were asked to treat a case of gastric ulcer or diabetes in one consultation.

The solution of these difficulties in hospital is that the honorary medical officer and the consultant should work together in constant and close cooperation.

The foregoing paragraphs are a summary of what I wrote in 1948. (The full report, revised in November 1955, is available). I have persisted in advocating the same principles—that in the children's wards the consultant, after seeing the patients in consultation, should be allowed to revisit at his discretion, advise investigation, and modify treatment *in partnership* with the medical officer in charge. The consultant's services should not be circumscribed or curtailed. To adhere to the narrowest meaning of 'consultant' is to attenuate the usefulness of the consultant. Most of the children are very ill and mal-nourished, and an isolated consultation without continuous contact with the case is not conductive to good medicine. Let us meet half-way and give each child admitted the benefit of cooperative scientific medicine, which should be the birthright of humanity.

A sincere and unprejudiced discussion and a firm decision can make the hospital an ideal place for the specialist to practice modern medical science in; it can become a haven of refuge for the general practitioner seeking to broaden his medical knowledge and to increase his usefulness to society. Such a hospital can become a great healing centre for the less fortunate of our population. Let us then abandon the narrow outlook and adopt the wider view. It has been conclusively shown that cooperative medicine, in sick children, shortens their stay in hospital. More children can be admitted per year and fewer turned away. This is worthy human conduct, good medicine, and sound medical economics.

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1. McMurray, T. B. (1956): S. Afr. Med. J., **30**, 296.